This is a digital copy of a book that was preserved for generations on library shelves before it was carefully scanned by Google as part of a project to make the world's books discoverable online.

It has survived long enough for the copyright to expire and the book to enter the public domain. A public domain book is one that was never subject to copyright or whose legal copyright term has expired. Whether a book is in the public domain may vary country to country. Public domain books are our gateways to the past, representing a wealth of history, culture and knowledge that's often difficult to discover.

Marks, notations and other marginalia present in the original volume will appear in this file - a reminder of this book's long journey from the publisher to a library and finally to you.

Usage guidelines

Google is proud to partner with libraries to digitize public domain materials and make them widely accessible. Public domain books belong to the public and we are merely their custodians. Nevertheless, this work is expensive, so in order to keep providing this resource, we have taken steps to prevent abuse by commercial parties, including placing technical restrictions on automated querying.

We also ask that you:

+ *Make non-commercial use of the files* We designed Google Book Search for use by individuals, and we request that you use these files for personal, non-commercial purposes.

+ *Refrain from automated querying* Do not send automated queries of any sort to Google's system: If you are conducting research on machine translation, optical character recognition or other areas where access to a large amount of text is helpful, please contact us. We encourage the use of public domain materials for these purposes and may be able to help.

+ *Maintain attribution* The Google "watermark" you see on each file is essential for informing people about this project and helping them find additional materials through Google Book Search. Please do not remove it.

+ *Keep it legal* Whatever your use, remember that you are responsible for ensuring that what you are doing is legal. Do not assume that just because we believe a book is in the public domain for users in the United States, that the work is also in the public domain for users in other countries. Whether a book is still in copyright varies from country to country, and we can't offer guidance on whether any specific use of any specific book is allowed. Please do not assume that a book's appearance in Google Book Search means it can be used in any manner anywhere in the world. Copyright infringement liability can be quite severe.

About Google Book Search

Google's mission is to organize the world's information and to make it universally accessible and useful. Google Book Search helps readers discover the world's books while helping authors and publishers reach new audiences. You can search through the full text of this book on the web at `http://books.google.com/`

LANE MEDICAL LIBRARY STANFORD
2 45 0425 8323

DISEASES OF THE HEART
AND
ARTERIAL SYSTEM

*DESIGNED TO BE A PRACTICAL PRESENTATION OF
THE SUBJECT FOR THE USE OF STUDENTS
AND PRACTITIONERS OF MEDICINE*

BY
ROBERT H. BABCOCK, A. M., M. D.
Professor of Clinical Medicine and Diseases of the Chest, College of Physicians and Surgeons (Medical Department of the Illinois State University), Chicago; Attending Physician to Cook County Hospital and Cook County Hospital for Consumptives; Consulting Physician to Mary Thompson Hospital, Hospital of St. Anthony de Padua, and of Marion-Sims Sanitarium; Fellow and former President of the American Climatological Association; Member of the American Medical Association, etc.

WITH THREE COLOURED PLATES AND
ONE HUNDRED AND THIRTY-NINE ILLUSTRATIONS

NEW YORK AND LONDON
D. APPLETON AND COMPANY
1903

Copyright, 1903
By D. APPLETON AND COMPANY

Published March, 1903

PRINTED AT THE APPLETON PRESS,
NEW YORK, U. S. A.

B112
1903

PREFACE

In the preparation of this work the author has endeavoured to present the subject in a simple, practical fashion that would suit the needs of the student and practitioner of medicine. Theories and speculations have been omitted or given but scanty consideration, in the belief that they tend to confuse the student. The anatomy and physiology of the circulatory organs have received only such notice as was thought necessary to a better understanding of the matter in hand, since an extended consideration of them was believed out of place in a work devoted to diseased conditions. Although aware that physical signs are properly a part of the symptomatology of disease and should be considered under that head, still the author has thought it best to consider them separately, for the sake of facilitating the knowledge of that most difficult subject, the diagnosis of cardiac disease. Special attention has been paid to treatment, and this part of the subject will be found far more detailed than is the case in most books dealing with diseases of the heart. It was hoped that by so doing the work might be given a more practical value to the general practitioner, although of course the author realized that he would lay himself open to adverse criticism, and could do but little more than lay down principles for management. The phraseology has been kept simple and free from needless technicalities, while in the terminology an attempt has been made to employ the terms which are in most familiar use among American and English physicians. No claim is laid to originality, as is apparent from the numerous references to authors from whose works valuable suggestions and information have been derived. To all such authors, grateful acknowledgment is made.

In conclusion the writer desires to express particular thanks to the following gentlemen: Drs. O. L. Schmidt, for the article on Gaertner's Tonometer; Edward F. Wells, for that on the Sphygmograph; Gustav Fütterer, for anatomical specimens and photographs; W. A. Evans, for post-mortem examinations and other aid; and Milton W. Hall, for preparing the illustrations. Finally, the author wishes to publicly express his indebtedness to his wife, for her encouragement to undertake this work, for her perusal of his manuscript, and suggestions, without which many passages might have been obscure, and for her invaluable aid in the revision of proof.

ROBERT H. BABCOCK.

103 STATE STREET, CHICAGO, ILL.

CONTENTS

GENERAL CONSIDERATIONS PERTAINING TO THE ANATOMY, PHYSIOLOGY, AND EXAMINATION OF THE HEART

PAGE
Introductory 1
Location of the heart 1
The relations of the heart to the anterior thoracic wall 2
Position of the great vessels and valves 3
Cardiac percussion 5
Auscultatory or stethoscopic percussion 8
Palpatory percussion 10
Auscultation of the heart 12
Normal heart-sounds 13
Reduplication of the heart-sounds 16
Reduplication of the first sound 18
Gallop or canter rhythm 18
Murmurs 21
Endocardial murmurs of organic origin 21
Cardiac areas 25
Accidental murmurs 26
Musical murmurs 29
Accidental musical murmurs 32
The differential diagnosis of accidental heart murmur 34
Exocardial murmurs 36

SECTION I

DISEASES OF THE PERICARDIUM

CHAPTER I

ACUTE PERICARDITIS

Morbid anatomy 37
Etiology 41

DRY PERICARDITIS

Symptoms 48
Course and termination 56

PAGE
Physical signs. *Inspection* 56
Palpation 57
Percussion 57
Auscultation 57
Location of the pericardial friction-sound 58
Rhythm of the friction-sound 58
Intensity of the friction-sound 59
Quality of the friction-sound 59
Effect of pressure on the pericardial murmur 59
Diagnosis 60
Differential diagnosis 60
Prognosis 61

PERICARDITIS WITH EFFUSION

Symptoms 65
Course and termination 73
Physical signs. *Inspection* 74
Palpation 75
Percussion 76
Auscultation 79
Secondary physical signs referable to the lungs 80
Diagnosis 81
Differential diagnosis 82
Prognosis 84
Treatment 86
Treatment in the stage of effusion 90

CHAPTER II

CHRONIC PERICARDITIS

Morbid anatomy 100
Etiology 103
Symptoms 104
Course and termination 117
Physical signs. *Inspection* 118
Palpation 120
Percussion 121
Auscultation 121
Diagnosis 122
Prognosis 123
Treatment 124

CHAPTER III

I. HYDROPERICARDIUM

Morbid anatomy 127
Etiology 128
Symptoms 128
Physical signs. *Inspection* 128
Palpation 128

PAGE
Percussion 129
Auscultation 129
Diagnosis 129
Prognosis 129
Treatment 130

II. HÆMOPERICARDIUM

Morbid anatomy 130
Etiology 130
Symptoms 131
Physical signs 131
Diagnosis 131
Prognosis 131
Treatment 131

III. PNEUMOPERICARDIUM

Morbid anatomy 132
Etiology 132
Symptoms 133
Physical signs. *Inspection* 134
Percussion 134
Auscultation 134
Diagnosis 135
Prognosis 135
Treatment 135

IV. TUBERCULOSIS OF THE PERICARDIUM

Morbid anatomy 136
Etiology 137
Symptoms 138
Physical signs 138
Diagnosis 138
Prognosis 138
Treatment 138

V. SYPHILIS OF THE PERICARDIUM

Morbid anatomy 139
Etiology 140
Symptoms 140
Physical signs 140
Diagnosis 141
Prognosis 141
Treatment 141

VI. CARCINOMA AND SARCOMA OF THE PERICARDIUM

Morbid anatomy 141
Etiology 142
Symptoms 142
Physical signs 142
Diagnosis 142
Prognosis and treatment 142

SECTION II

DISEASES OF THE ENDOCARDIUM

CHAPTER IV

ACUTE ENDOCARDITIS

PAGE
Morbid anatomy 144
Etiology 150
SIMPLE ENDOCARDITIS 152
ULCERATIVE ENDOCARDITIS 155
Symptoms 157
ACUTE SIMPLE ENDOCARDITIS 157
Diagnosis 163
Course and termination 163
Ulcerative endocarditis 163
Course and termination 172
Physical signs. *Inspection* 176
Palpation 176
Percussion 177
Auscultation 177
Diagnosis 178
Diagnosis of ulcerative endocarditis 179
Prognosis 183
Treatment 187
Treatment of acute ulcerative endocarditis 191

CHAPTER V

CHRONIC ENDOCARDITIS

Morbid anatomy 199
Etiology 201
Symptoms 205

CHAPTER VI

MITRAL REGURGITATION

Morbid anatomy 216
Etiology 221
Symptoms 223
Physical signs. *Inspection* 239
Palpation 239
Percussion 240
Auscultation 242
Diagnosis 245
Prognosis 246
Mode and causes of death 247

CHAPTER VII

MITRAL STENOSIS

Morbid anatomy 249
Etiology 252

PAGE
Symptoms 255
Physical signs. *Inspection* 258
Palpation 259
Percussion 260
Auscultation 261
Diagnosis 268
Prognosis 269
Mode and causes of death 270

CHAPTER VIII

AORTIC REGURGITATION

Morbid anatomy 278
Etiology 280
Symptoms 282
Physical signs. *Inspection* 297
Palpation 298
Percussion 301
Diagnosis 305
Prognosis 306
Mode and causes of death 307

CHAPTER IX

AORTIC STENOSIS

Morbid anatomy 319
Etiology 322
Symptoms 323
Physical signs. *Inspection* 335
Palpation 335
Percussion 336
Auscultation 337
Diagnosis 338
Prognosis 339
Mode and causes of death 340

CHAPTER X

TRICUSPID REGURGITATION

Morbid anatomy 344
Etiology 345
Symptoms 347
Physical signs. *Inspection* 349
Palpation 350
Percussion 351
Diagnosis 353
Prognosis 354
Mode and causes of death 354

CHAPTER XI

TRICUSPID STENOSIS

Morbid anatomy 355
Etiology 356

PAGE
Symptoms 357
Physical signs. *Inspection* 361
Percussion 362
Auscultation 362
Diagnosis 363
Prognosis 364
Mode and causes of death 364

CHAPTER XII

PULMONARY REGURGITATION

Morbid anatomy 365
Etiology 366
Symptoms 367
Physical signs 370
Inspection 371
Palpation 371
Percussion 371
Auscultation 372
Diagnosis 373
Prognosis 374
Mode and causes of death 374

CHAPTER XIII

PULMONARY STENOSIS

Morbid anatomy 376
Etiology 380
Symptoms 380
Physical signs. *Inspection* 385
Palpation 386
Percussion 386
Auscultation 386
Diagnosis 387
Prognosis 387
Mode and causes of death 388
Summary of physical signs of valve lesions of the right heart 389

CHAPTER XIV

COMBINED VALVULAR LESIONS

Combined mitral stenosis and regurgitation 390
  Symptoms 391
  Diagnosis 391
  Prognosis 392
MITRAL STENOSIS AND AORTIC STENOSIS 392
  Symptoms 392
  Diagnosis 392
  Prognosis 393

PAGE
MITRAL STENOSIS AND AORTIC REGURGITATION 393
Symptoms 393
Diagnosis 394
Inspection 395
Palpation 395
Percussion 395
Auscultation 395
Prognosis 396
MITRAL REGURGITATION AND AORTIC STENOSIS 396
Symptoms 396
Diagnosis 396
Prognosis 396
AORTIC REGURGITATION AND MITRAL REGURGITATION 397
Symptoms 397
Diagnosis 397
Prognosis 398
AORTIC STENOSIS AND AORTIC REGURGITATION 398
Symptoms 398
Physical signs 399
Diagnosis 399
Prognosis 400

CHAPTER XV

THE PROGNOSIS OF VALVULAR HEART-DISEASE IN GENERAL

Complications 405
Rheumatic diathesis 406
Digestive and bronchial disorders 407
Age 407
Temperament 408
Sex 409
Occupation 409
Habits 410
Home surroundings 410
The probable effect on the patient of the knowledge of his lesion 411
The effect of digitalis on the patient 411
The relation of prognosis to life insurance 412

CHAPTER XVI

THE TREATMENT OF VALVULAR HEART-DISEASE

I. Compensation being still perfect 414
Exercise 414
Occupation 419
Habits 420
Marriage 422
Clothing 425
Baths 427
Food 428
Illnesses 429
Use of drugs 430
Change of climate, with special reference to high altitude 432

CHAPTER XVII

THE TREATMENT OF VALVULAR HEART-DISEASE (*continued*)

PAGE
II. Compensation being imperfect 435
Medicinal agents 444
Rest 448
Exercise 454
Resistance exercise 455
Nauheim baths 464
Diet 470
Clothing, habits, occupation 476

CHAPTER XVIII

THE TREATMENT OF VALVULAR HEART-DISEASE (*concluded*)

III. Compensation lost 478
The treatment of dropsy 489
Cathartics 492
The use of digitalis 494
Accessory heart tonics 499
Hypnotics 500
Rest 502
Exercise 502
Baths 503
Receiving visitors 503
Diet 503

SECTION III

DISEASES OF THE MYOCARDIUM

CHAPTER XIX

ACUTE MYOCARDITIS

Morbid anatomy 506
Etiology 508
Symptoms 510
Physical signs. *Inspection* 514
Palpation 514
Percussion 514
Auscultation 514
Diagnosis 514
Prognosis 515
Treatment 515

CHAPTER XX

CHRONIC MYOCARDITIS

Morbid anatomy 519
Etiology 522

PAGE
Symptoms 526
Physical signs. *Inspection* 543
Palpation 543
Percussion 544
Auscultation 545
Diagnosis 547
Prognosis 549
Treatment 551
Commencing loss of heart-power 553
Cardiac incompetency pronounced 555

CHAPTER XXI

HYPERTROPHY OF THE HEART

Morbid anatomy 565
Etiology 568
Symptoms 570
Physical signs. *Inspection* 571
Palpation 571
Percussion 571
Auscultation 572
Diagnosis 572
Prognosis 574
Treatment 575

CHAPTER XXII

DILATATION OF THE HEART—RELATIVE MITRAL INSUFFICIENCY

I. DILATATION OF THE HEART

Morbid anatomy 576
Etiology 577
Symptoms 580
Physical signs. *Inspection* 585
Palpation 585
Percussion 585
Auscultation 586
Diagnosis 586
Prognosis 587
Treatment 590
(1) *Bloodletting* 591
(2) *Nauheim baths* 592
(3) *Resistance exercises* 592

II. RELATIVE MITRAL INSUFFICIENCY

Pathology 594
Etiology 594
Symptoms 596
Physical signs and diagnosis 596
Prognosis 597
Treatment 597

CHAPTER XXIII

FATTY HEART—CARDIAC INADEQUACY OF THE CORPULENT

PAGE
Morbid anatomy 599
Pathology 599
Etiology 600
Symptoms 602
Physical signs. *Inspection* 604
Palpation 604
Percussion 604
Auscultation 605
Diagnosis 605
Prognosis 606
Treatment 606

CHAPTER XXIV

CARDIAC ASTHMA—CHEYNE-STOKES RESPIRATION—BRADYCARDIA—STOKES-ADAMS DISEASE

I. Cardiac asthma 613
II. Cheyne-Stokes respiration 615
  Diseases in which Cheyne-Stokes breathing is observed 617
  Theories to explain Cheyne-Stokes respiration 617
  Prognosis 622
  Treatment 623
III. Bradycardia 624
IV. Stokes-Adams disease 627
  Etiology and pathology 627
  Symptoms 629
  Prognosis 635
  Treatment 635

CHAPTER XXV

ANGINA PECTORIS

Definition 637
History 637
Pathology and etiology 640
Clinical history and features of an attack 649
Diagnosis 654
Prognosis 657
Treatment 658

CHAPTER XXVI

SYPHILIS OF THE MYOCARDIUM—NEW GROWTHS IN THE MYOCARDIUM—ATROPHY OF THE HEART—SEGMENTATION AND FRAGMENTATION OF THE MYOCARDIUM

I. Syphilis of the myocardium 663
  Morbid anatomy 663
  Etiology 663
  Symptoms 664
  Diagnosis 664

PAGE
Prognosis 665
Treatment 665
II. New growths in the myocardium 666
III. Atrophy of the heart 667
Morbid anatomy 667
Etiology 667
Symptoms. 668
Diagnosis 668
Prognosis 668
Treatment 668
IV. Segmentation and fragmentation of the myocardium 668

CHAPTER XXVII

PEDUNCULATED AND BALL THROMBI OF THE HEART

Pathogenesis and etiology 674
Symptoms 675
Diagnosis 677
Prognosis 678
Treatment 678
Bibliography of cases of ball thrombi 680

CHAPTER XXVIII

DEXTROCARDIA

Congenital dextrocardia 681
Symptoms. 681
Diagnosis 682
Acquired dextrocardia 682
Morbid anatomy 682
Etiology 683
Symptoms. 684
Diagnosis 684
Inspection and palpation 684
Percussion 684
Auscultation 684
Prognosis 685
Treatment 685

CHAPTER XXIX

CONGENITAL DISEASES OF THE HEART

Morbid anatomy 686
Etiology 689
Symptoms 690
Physical signs. *Inspection*. 695
Palpation 696
Percussion 697
Diagnosis 701
Prognosis 701
Treatment 702

SECTION IV

CARDIAC NEUROSES

Syn.: *Functional Disorders of the Heart*

CHAPTER XXX

PALPITATION, TACHYCARDIA, CARDIAC PAIN, PSEUDO-ANGINA PECTORIS

PAGE

Pathology 703
Symptoms 704
Palpitation 704
Tachycardia. 715
Cardiac pain 718
Pseudo-angina pectoris 719
Etiology 722
Diagnosis 724
Prognosis 726
Treatment 727
Treatment of the attack 727
Palpitation 727
The attack of pain 728

CHAPTER XXXI

ESSENTIAL PAROXYSMAL TACHYCARDIA

Pathology 731
Etiology 732
Features of the paroxysm 732
Diagnosis 734
Prognosis 735
Treatment 735

SECTION V

DISEASES OF THE ARTERIAL SYSTEM

CHAPTER XXXII

ARTERIOSCLEROSIS 738
Morbid anatomy 739
Etiology 741
Symptoms 745
Physical signs 750
Diagnosis 751
Prognosis 754
Treatment 754

CHAPTER XXXIII

ACUTE AORTITIS — ACUTE ARTERITIS — SYPHILITIC ARTERITIS — ENDARTERITIS OBLITERANS—PERIARTERITIS NODOSA—STENOSIS OF THE AORTA AND PULMONARY ARTERY—CONGENITAL SMALLNESS OF THE ARTERIES

I. ACUTE AORTITIS

PAGE
Morbid anatomy 759
Etiology 760
Symptoms 760
Physical signs 761
Inspection 761
Palpation 761
Percussion 761
Auscultation 762
Diagnosis 762
Prognosis 762
Treatment 762

II. ACUTE ARTERITIS

Morbid anatomy 762
Symptoms 763
Physical signs. *Inspection—Palpation* 763
Diagnosis 763
Prognosis 763
Treatment 764

III. SYPHILITIC ARTERITIS

Morbid anatomy 764
Etiology 765
Symptoms 765
Diagnosis 766
Prognosis 766
Treatment 766

IV. ENDARTERITIS OBLITERANS

Morbid anatomy 766
Etiology 767
Symptoms 767
Diagnosis 768
Prognosis and treatment 769

V. PERIARTERITIS NODOSA

SYN.: *Congenital Aneurysm*

Morbid anatomy 769
Etiology 769
Symptoms 769
Diagnosis 770
Prognosis and treatment 770

VI. STENOSIS OF THE AORTA AND PULMONARY ARTERY

PAGE

Stenosis of the aorta, congenital and acquired 770
  Symptoms 771
  Diagnosis 771
  Prognosis and treatment 772
Stenosis of the pulmonary artery 772
  Symptoms 772
  Diagnosis 772
  Prognosis and treatment 773

VII. CONGENITAL SMALLNESS OF THE ARTERIES

Symptoms 773
Diagnosis 774
Prognosis and treatment 774

CHAPTER XXXIV

ANEURYSM OF THE THORACIC AORTA

Morbid anatomy 775
Etiology 777
Symptoms 781
Pain 782
Dyspnœa 783
Cough 784
Expectoration 784
Physical signs. *Inspection*. 800
Palpation 801
Percussion 802
Auscultation 802
Diagnosis 804
Prognosis 808
Modes and causes of death 808
Treatment 809

APPENDIX

MECHANICAL DEVICES AS AIDS TO DETERMINING CARDIAC DISEASE . . . 815
The X-ray 815
The sphygmograph 818
Gaertner's tonometer 826

LIST OF PLATES AND ILLUSTRATIONS

PLATE FACING PAGE

I. Anatomical relations of thoracic and abdominal viscera 1
II. Aortic regurgitation with calcified vegetation that swung in blood current, causing atheroma of endocardium and of intima of aorta . . 278
III. Exterior of heart of Fig. 42, showing hypertrophy and dilatation of both ventricles 576

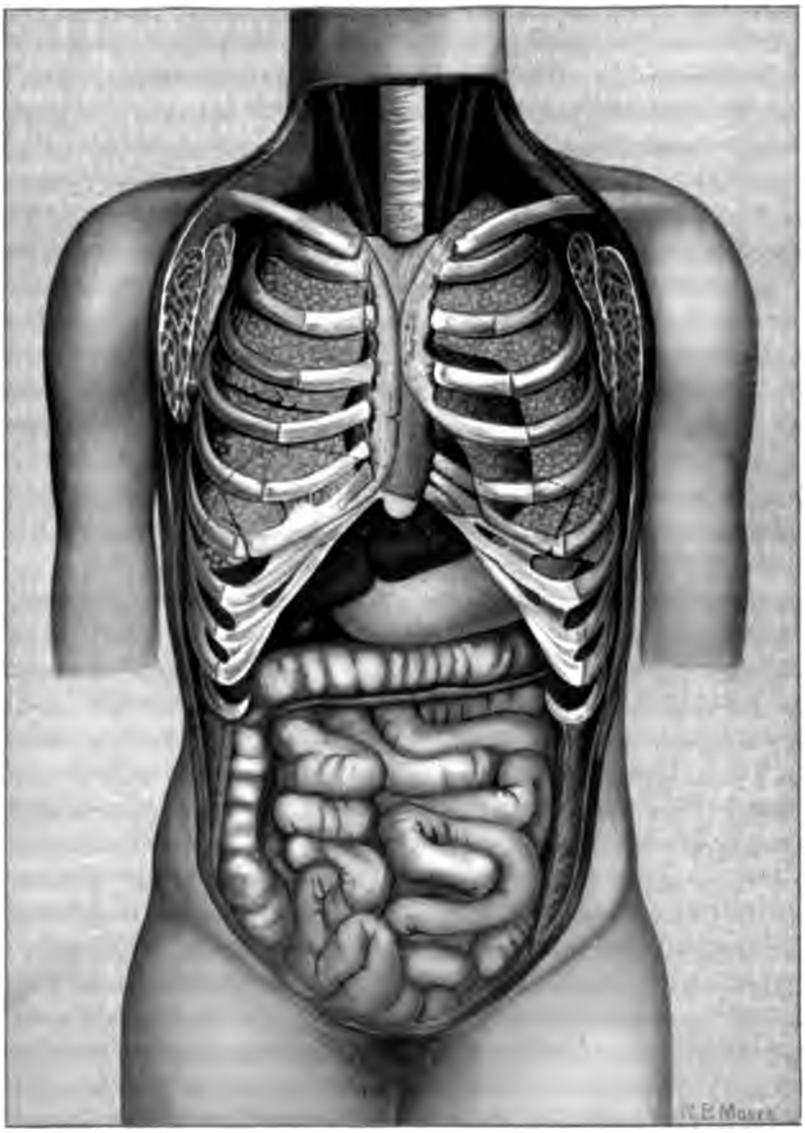
PAGE

Cardiac valve areas, *showing where sounds are most distinctly heard* . . 4
Normal deep-seated cardiac dulness 6
Auscultatory percussion 8, 9
Hein's palpatory percussion 10, 11
Ebstein's palpatory percussion 11
Maguire's palpatory percussion 12
Normal cardiac cycle 14
Cardiac valve areas, *indicating sounds produced at various valves* . . . 25
Interior of left ventricle, *showing fibrous band connecting aortic cusps and responsible for musical murmur* 31
Heart of a buffalo calf, *showing aberrant chordæ tendinæ in left ventricle* . 33
Cor villosum of acute plastic pericarditis 39
Usual location of pericardial friction sound and fremitus 58
Absolute dulness in case of acute pericarditis 63
Case of pericarditis in which the sac contained 3½ pounds of fluid (Bramwell) 65
Absolute dulness in case of pericarditis with effusion 71
Rotch's sign of beginning effusion 78
Pins and Ewart's signs of pericardial effusion 80
Apex-beat and area of cardiac dulness in case of pericarditis with effusion . 93
The various sites for puncture in paracentesis pericardii 94
Cardiac dulness and location of border of liver in special cases . . 108, 115
Verrucose endocarditis of aortic and mitral valves 145
Verrucose endocarditis 146
Malignant verrucose endocarditis of mitral valve 147
Malignant verrucose endocarditis of aortic valve, with perforation of a cusp . 149
Apex-beat and relative dulness in case of acute endocarditis 159
Apex-beat and absolute dulness later in same case 161
Apex-beat and relative cardiac dulness; *special case* 165
Area of maximum audibility and transmission of murmur; *special case* . 165
Chart I. Temperature in case of acute endocarditis 167
Chart II. Temperature in case of acute endocarditis 173
Diminution of relative cardiac dulness in one week under treatment . . 186
Relative dulness in case of chronic endocarditis 203
Condition of mitral valve causing regurgitation and obstruction . . . 217

PAGE
Diagram showing effects of a mitral leak on the circulation 220
Relative dulness in case of mitral insufficiency 227
Apex-beat and relative dulness in mitral regurgitation 230
Sphygmogram, showing irregular pulse in case of mitral regurgitation . . 240
Relative dulness in a typical case of mitral regurgitation 241
Point of maximum audibility and area of transmission of mitral regurgitant murmur 242
Time of mitral regurgitant murmur 243
Interior of left ventricle, showing buttonhole slit 250
Case of mitral stenosis, showing ascites and clubbing of finger-tips . . 258
Sphygmogram, from case of mitral stenosis 260
Location of apex-beat and area of deep-seated dulness in mitral stenosis . 260
Rhythm of characteristic murmur of mitral stenosis, "auricular systolic" . 261
Area of audibility of the presystolic murmur of mitral stenosis . . . 262
Rhythm of occasional variety of mitral stenotic murmur through entire ventricular diastole 264
"Interrupted modified presystolic" murmur of mitral stenosis . . . 265
Location of apex and relative dulness in case of mitral stenosis . . . 271
Location of apex and relative dulness in case of mitral stenosis and regurgitation 273
Location of apex and relative dulness in case of mitral stenosis . . . 276
Location of apex and relative dulness in case of aortic regurgitation . . 285
Sphygmogram of aortic regurgitation 299
Sphygmogram of pulsus bisferiens 300
Type of relative dulness in well-compensated aortic regurgitation . . . 302
Type of relative dulness in poorly compensated aortic regurgitation . . 302
Spot of maximum intensity and area of transmission of typical aortic regurgitant murmur 303
Rhythm of aortic regurgitant murmur 303
Relative dulness in case of aortic regurgitation 311
Skiagram of chest in case of aortic regurgitation 312
Relative dulness and lower border of liver shortly before death . . . 316
Heart of aortic stenosis with adherent cusps and also acute endocarditis . 320
Heart of aortic stenosis showing calcified vegetations in sinuses of Valsalva . 321
Sphygmogram from case of aortic stenosis 329
Sphygmogram of uncomplicated aortic stenosis 336
Typical relative dulness in case of well-compensated aortic stenosis . . 336
Rhythm of aortic obstructive murmur 337
Place of maximum intensity and propagation of aortic stenotic murmur . 337
Relative dulness in case of primary tricuspid regurgitation 351
Relative dulness in case of tricuspid regurgitation secondary to dilatation of right ventricle 352
Place of maximum audibility and area of propagation of tricuspid regurgitant murmur 352
Location of thrill and murmur in a typical case of tricuspid stenosis . . 361
Relative cardiac dulness in typical case of tricuspid stenosis 362
Area of deep-seated cardiac dulness in case of pulmonary regurgitation . . 368
Area of maximum intensity and of propagation of murmur in case of pulmonary regurgitation 369
The rhythm of murmur in typical case of pulmonary regurgitation . . 373
Heart of a boy, showing congenital stenosis of the pulmonary orifice . . 379

PAGE
Relative cardiac dulness in case of pulmonary stenosis 381
Location of thrill and systolic murmur in case of pulmonary stenosis . . 381
Heart from case of pulmonary stenosis 383
Same heart, left auricle open, showing patent foramen ovale 384
Rhythm of typical pulmonary stenotic murmur 387
Resistance exercises 456, 457, 460–463
Shape of relative dulness in hypertrophy 543
Heart showing left ventricular hypertrophy 566
Perforate interventricular sæptum 687
Cut showing cyanosis of congenital heart-disease, drum-stick finger-tips, bulging præcordia, etc. 691
Heart showing concentric hypertrophy of left ventricle 699
Sphygmogram case of paroxysmal tachycardia 733
Skiagraph showing aneurysm of the aorta 778
Dilatation of superficial veins secondary to pressure by aneurysm on venæ cavæ 786
Photograph: aortic aneurysm, showing slight bulging of anterior chest-wall. 788
Cut showing dulness and liver outline in case of aneurysm 789
Two figures showing external tumour in case of aortic aneurysm . . . 790
Post-mortem specimen of heart and aneurysmal sac 791
Trachea, from case of ruptured aneurysm, showing point of rupture . . 797
Opposite side of same specimen, showing interior of sac 798
Skiagraph of chest, showing tuberculosis of right apex and tuberculous pericarditis with effusion 817
Sphygmogram of healthy man 819
Sphygmogram of woman during an attack of paroxysmal tachycardia . . 819
  From man with recurrent bradycardia 819
  From man with acute general gonorrhœal infection 820
  From man with declining typhoid fever 820
Hyperdicrotic pulse from woman after twelve hours recurring hæmoptysis . 820
  From woman with moderate aortic insufficiency well compensated . . 821
Initial high-tension pulse from man with arteriosclerosis 821
Sustained high-tension pulse from woman with chronic interstitial nephritis. 821
  From a man with chronic interstitial nephritis 822
  From a woman with mild myxœdema 822
  From a man with well-compensated mitral iusufficiency 822
  From a woman with arteriosclerosis and fairly well-compensated mitral incompetence 823
  From a woman with arteriosclerosis and mitral insufficiency fairly compensated 823
  From a man with arteriosclerosis, chronic interstitial nephritis, and mitral insufficiency, with failing compensation, Cheyne-Stokes respiration . . 824
  From a woman with mitral obstruction and regurgitation with failing compensation 824
  From a man with mitral regurgitation, lost compensation, relative tricuspid insufficiency, ascites, etc. 824
  From a woman with mitral obstruction and insufficiency, lost compensation and relative incompetence of the tricuspid. Delirium cordis . . . 824
  From a boy with acute rheumatism on second day of endocarditis . . 825
  From same patient, two years later, with developed aortic stenosis . . 825
Cardiogram from a girl with mitral insufficiency 826

PLATE I



ANATOMICAL RELATIONS OF THORACIC AND ABDOMINAL VISCERA.

DISEASES OF THE HEART

GENERAL CONSIDERATIONS PERTAINING TO THE ANATOMY, PHYSIOLOGY, AND EXAMINATION OF THE HEART

INTRODUCTORY

In this chapter are presented certain facts which, because of their bearing on the examination and the diseases of the heart, should be well understood.

Location of the Heart.—This main organ of circulation is situated in the central and lower part of the thoracic cavity, resting upon the upper convex surface of the diaphragm (see frontispiece) in such a manner that its long axis forms an angle of sixty degrees with that of the body (Rosenstein). The base of the organ is directed upward, backward, and towards the right side, while its apex points downward, forward, and to the left, so as to strike against the chest-wall in the fifth left intercostal space an inch inside the nipple-line. The larger portion of the heart, therefore, lies to the left of the median line. It is attached at its base to the great vessels and is inclosed by the pericardial sac, which invests it loosely below, being bound to the central tendon of the diaphragm beneath, to the sternum in front, to the mediastinal pleura at each side, and behind to the anterior surface of the œsophagus, trachea, and large bronchi. In consequence of the oblique position of the heart, the pericardial sac forms a loose fold at the lower right-hand corner, so that when it becomes distended by an effusion the earliest evidence of the fluid is obtained in the fifth right interspace in what is known as the *cardio-hepatic angle.* Further details on this matter are contained in the chapter on Acute Pericarditis.

The Relations of the Heart to the Anterior Thoracic Wall are highly important, and have been the subject of numerous investigations. As it is difficult to fix the heart in position for the purpose of investigation, attempts have been made to accomplish this either by thrusting long needles through the organ immediately after death or by freezing the cadaver, and subsequently sawing it into sections. Consequently the statements of investigators differ somewhat as to the limits of the heart in health, especially the level of its upper border. I shall state the lower of the two levels usually given, therefore, as it seems to me to correspond with what is most often observed clinically.

The *superior boundary* of the heart lies on a level with the upper border of the third costal cartilages and extends transversely from the third left costo-chondral articulation across the sternum to a point about an inch to the right of the right sternal margin. The *right border* of the heart, formed by the slightly convex base of the right auricle, extends from the upper edge of the third right costal cartilage, at the point where the superior cardiac boundary ceases, downward in a slightly convex direction as far as the middle of the fifth right interspace, about an inch from the breastbone. Here, curving sharply inward, the *inferior border* of the organ passes across the base of the xiphoid process on a level with the upper margin of the sixth left costal cartilage at its junction with the sternum and terminates in the fifth left interspace at the site of the apex-beat, an inch inside the nipple-line. The *left cardiac border* corresponds to a line drawn from the apex-beat upward and somewhat inward to the junction of the third left rib with its cartilage, about 2 inches from the left sternal margin. A diagonal line extending from the junction of the third costal cartilage with the left edge of the sternum downward to the seventh right chondro-sternal articulation, represents the usual position of the auriculo-ventricular sæptum (Broadbent). A line which passes from the inner side of the apex upward through the fifth and fourth to the third left costo-chondral articulation, corresponds as closely as can be with the interventricular sæptum. The somewhat triangular area thus inclosed represents the right ventricle with its broad base forming the lower boundary of the heart, which rests in the sulcus between the anterior chest-wall and the upper surface of the diaphragm. The upper extremity of this triangular area is filled by the pulmonary artery and the tip of the left auricular appendix as it curves around the outer border of the left ventricle to appear in front to and terminate near the trunk of the great artery. It is obvious, therefore, that only the upper third of the right auricle lies behind the sternum, while its lower two thirds are to the right of this bone. The left auricle is situated behind, being completely invested by the left lung and entirely obscured from view from the front. The same is the case also with the left ventricle, excepting a narrow strip which forms the left border of the heart and is visible anteriorly. It is the inferior extremity of this narrow strip which, propelled against the wall of the thorax, occasions the apex-beat. Consequently it is a portion of the right ventricle only which is exposed to view after removal of the breastbone and adjacent costal cartilages. The remainder of the heart, even that which lies anteriorly, is covered from view by the lungs.

The anterior lung borders are in apposition behind the middle of the sternum from the level of the second to that of the fourth costal cartilages. At this latter situation they diverge, the border of the right lung passing on downward to the level of the fifth right costal cartilage, where it turns off to the right to unite with the inferior margin of the same lung. The anterior margin of the left lung diverges abruptly at the level of the fourth cartilage, passing outward along the lower edge of this cartilage as far as its union with its rib. It then turns downward, and, after curving slightly inward and then outward, unites with the inferior border at the level of the sixth costal cartilage near its point of articulation with its rib. In consequence of this peculiar arrangement of the left lung a portion of the anterior surface of the right ventricle comes into immediate contact with the chest-wall, and, being uncovered by lung, forms the *area of superficial cardiac dulness.* By many this area is considered of great importance in the determination of the size of the heart by percussion, as will be shown in dealing with the subject of cardiac percussion.

Position of the Great Vessels and Valves.—The pulmonary artery lies about half an inch to the left of the breastbone and extends from the level of the centre of the third left interspace upward to the level of the second costal cartilage, where it divides into its two main branches. The position and course of the ascending aorta may be represented by a line drawn from the third left chondro-sternal articulation upward across the breastbone to the junction of the right edge of that bone with the second right costal cartilage, which, therefore, is sometimes spoken of as the aortic cartilage, because at this point the aortic valve-sounds are most distinctly heard. The superior vena cava passes downward along the right cardiac border from the level of the second costal cartilage to a point opposite the middle of the third right interspace.

The four sets of valves are bunched closely together not far from the junction of the third left costal cartilage with the border of the sternum, the pulmonary being most superficial, the mitral most internal, the tricuspid most inferior, and the aortic the most central. They cannot, therefore, be auscultated in the region of their anatomic seat if one is to differentiate their individual sounds. For this reason we take advantage of the laws governing the conduction of their sounds and auscultate them in certain areas named after the respective valves.

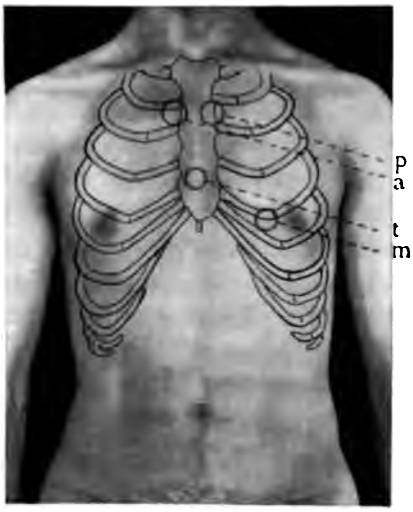


Fig. 1.—Cardiac Valve Areas.

Sounds produced at various valves indicated: *p*, pulmonary; *a*, aortic; *t*, tricuspid; *m*, mitral.

Thus the *mitral area* is situated at the apex-beat and includes a limited district immediately roundabout. The *tricuspid area* includes the lower end of the sternum and a portion of the surrounding region. The *pulmonic area* is located in the second left intercostal space close to the edge of the breastbone, while the *aortic area* lies in the corresponding situation on the opposite side. It must not be supposed that the valve-sounds and murmurs are heard only in these situations—they are widely propagated and blend with one another, and in particular endocardial murmurs are often so widely conducted as to be distinctly audible in other areas than those to which they properly belong.

Leaving further consideration of this subject at this time, we now pass on to the discussion of the methods by which the size of the heart is ascertained during life (Fig. 1).

Cardiac Percussion.—In employing this means of examination we aim to determine, first, the boundaries of the area of superficial dulness, and second, the limits of deep-seated dulness. To accomplish the former, percussion must be made lightly, whereas the latter requires a firm, heavy percussion-stroke.

The *area of superficial or absolute cardiac dulness* corresponding with the portion of the right ventricle uncovered by lung during inspiration, extends vertically from the upper edge of the fourth left costal cartilage to the sixth, and transversely from the left border of the sternum to a point midway between the parasternal and mamillary lines. As its outer or left boundary is irregular, and, roughly speaking, passes obliquely downward towards the left, this area is broader at its lower than at its upper margin. Enlargement of the heart crowds the lung-borders aside, and hence generally increases the dimensions of superficial dulness, especially to the right in cases of hypertrophy and dilatation of the right ventricle. But a variety of conditions outside of the heart may increase or diminish the extent of superficial dulness, and hence render this not always a trustworthy indication of the actual size of the heart. Thus the lung-borders may be retracted by pleuritic adhesions and expose an abnormally large portion of the right ventricle, or being distended by pulmonary emphysema, they may diminish or entirely obliterate this area.

Consequently it is preferable to rely upon deep rather than superficial percussion in endeavouring to ascertain the size of the heart, since when the limits of deep-seated or relative cardiac dulness are found increased we know it is due to increase in the size of the organ itself. Vierordt objects to this latter method because of its greater difficulty and uncertainty, since pulmonary resonance shades so gradually into the relative dulness overlying the heart that two observers of apparently equal skill may not agree in their results. Doubtless individual judgment depends very largely upon practice and delicacy of hearing, and doubtless emphysema, inelasticity of the ribs, great thickness of the parietes, etc., often make it impossible to accurately determine deep cardiac limits. Nevertheless the cases in which relative dulness is possible of detection are so numerous that I prefer to rely upon it rather than on superficial dulness, and always urge students to make use of this method.

The Deep Boundaries of the Heart (Fig. 2).—It is well known that all hearts are not of the same size even in health, the male heart being larger than the female, and that of a child relatively larger than that of an adult. Moreover, the right auricle measures more during diastole than during systole. Consequently measurements cannot be given that are invariable. Yet the following figures taken from Vierordt may be stated as the average. The adult heart "extends from about 8 or 9 centimetres to the left of the median line (apex of the heart) to about 4 or 5 centimetres to the right of the same, i. e., about one and a half finger-breadths to the right of the right border of the sternum (right auricle)." Busse, who employed Ebstein's palpatory percussion, found the left border of the heart in health never passed outside the mamillary line, while Hornkohl determined the average in adults to be 7.3 centimetres from the left sternal margin. On the right side the heart extended a variable distance beyond the sternum, depending on the stature of the man, being 2.0 centimetres for one 130 centimetres tall, and 3.0 centimetres for a male of 190 centimetres in height. In women these figures are slightly less, while in children the area of the heart measures relatively more than in adults. If the median line is taken as the landmark from which to measure, Hornkohl's figures must be increased by 1 to 1.5 centimetre, which, according to Ebstein, is half the width of the sternum. Consequently it is found that Vierordt's and Hornkohl's estimates are not so much at variance as they at first appear.

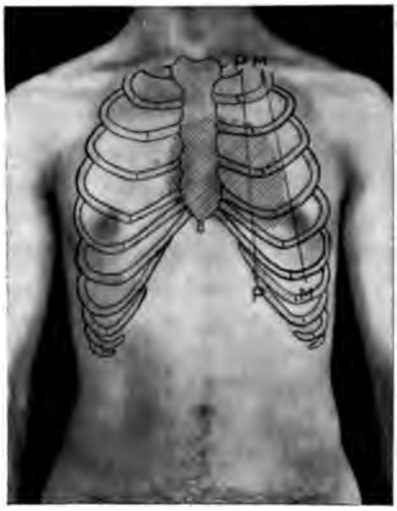


FIG. 2.—NORMAL DEEP-SEATED CARDIAC DULNESS.

PP, parasternal line; *MM*, mamillary line.

Three methods of percussion are in use, and mentioned in the order of their popularity are: (1) plessimetric, (2) auscultatory, (3) palpatory percussion. I do not propose to discuss the advantages or disadvantages of employing a pleximeter and hammer, but merely to express my very positive preference for the use of the fingers, for the reason that thereby one is enabled to obtain valuable information from the sense of resistance.

In ascertaining the area of absolute dulness light strokes are essential, while the reverse is the case as regards deep-seated dulness. Moreover, in outlining the area of relative dulness the pleximeter finger should be pressed firmly against the chest-wall, to exclude so far as possible the vibrations of the bony structures. This is the "*abgedämpfte*" percussion of the Germans. The finger is placed firmly at right angle to the ribs at a point well outside the cardiac area, and percussion is made with considerable force at ever decreasing distances from the sternum until a slight rise in pitch and increase of resistance indicate that the airless organ (the heart) has been reached.

In this manner one is to percuss from above downward along the left parasternal line, beginning in the first intercostal space and ceasing when the flatness of the liver has been reached. At the sides, percussion is to be performed first in an oblique direction from above downward and inward, and next on a transverse line from without towards the centre. If, wherever comparative dulness is perceived, a mark is made with a dermographic pencil, these marks can subsequently be united, and will then represent the probable limits and shape of deep-seated cardiac dulness. If one prefers he can, instead of placing his finger across the ribs, press it strongly into the intercostal space parallel with the ribs, and if his finger is slender can thus convey his percussion-strokes more directly to the heart without eliciting so much vibration from the elastic structures intervening.

Sansom makes use of a narrow pleximeter, which is of such small size as to fit well down into the intercostal space, and claims remarkably accurate results, more precise indeed than in any other way.

It may be well to here remark that, when in women accurate percussion of the heart is impossible on account of the large size of the mammæ, fairly trustworthy information concerning the size of the heart may be gained by careful palpation of the apex-beat. Since the mamillary line is not a trustworthy guide in females, it is better to measure the site of the apex impulse from the mid-sternal line or from the mid-clavicular line, it being in the fifth interspace, an inch within the latter.

Two statements should also be made regarding percussion of the heart in children. In the first place, the area of superficial dulness is said by Hornkohl to be somewhat more extensive than in adults, particularly above, where it is asserted to reach up into the third intercostal space, while its outer margin passes somewhat further beyond the left parasternal line, i. e., to a point a little nearer the mamillary than the parasternal line. In the second place, it is important to bear in mind the great elasticity of the child's chest, and hence to percuss with far more delicacy than is advisable in grown people. Otherwise the note of pulmonary resonance and the vibrations of underlying structures will assuredly prevent accurate and trustworthy results. For these reasons it is far preferable to rely on the other modes of percussion now to be described.

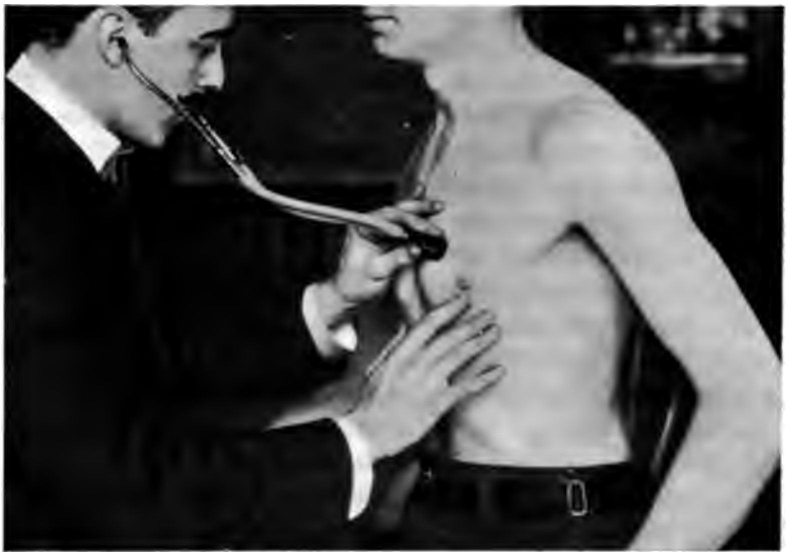


FIG. 3.—AUSCULTATORY PERCUSSION.

Auscultatory or Stethoscopic Percussion.—This is a combination of auscultation and percussion, and is based on the principle that when the stroke is made over a solid organ its note is higher, sharper, and more clearly defined than when over an air-containing organ. It is found, moreover, that there is a distinct difference in the character of the note of two viscera of similar structure. This is, of course, the same principle that underlies plessimetric percussion, but the auscultatory method enables one to appreciate more delicate shadings of tone and to define more precisely the deeply situated borders of an organ or solid thoracic tumour. It even enables one to distinguish between the dulness of pleuritic or pericardial effusion and that of a contiguous pulmonary consolidation.

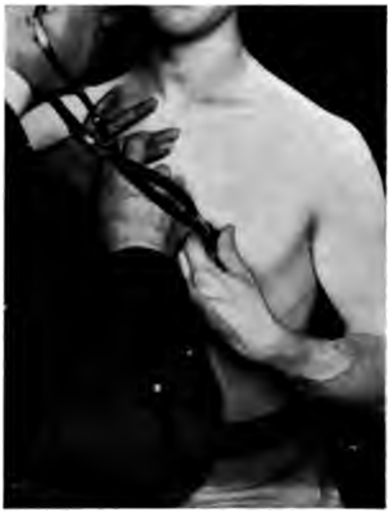


FIG. 4.—AUSCULTATORY PERCUSSION.

It is practised in either of two ways: The examiner may with one hand hold the bell of his binaural stethoscope against the centre of the cardiac area, while with the tip of a finger of the disengaged hand he taps the chest-wall lightly from without inward and on a line with his stethoscope (Fig. 3), or he may have his instrument held by an assistant while he performs percussion in the ordinary manner (Fig. 4). The former mode is preferable, because more delicate. Such astonishing and incredible accuracy is claimed for auscultatory percussion, notably by Bezly Thorne, that Broadbent and others have been led to test it, and have come to the conclusion that it possesses no advantages over plessimetric percussion. I have employed it a great deal, and, although recognising its liability to error and its limitations, still I believe it is in certain cases with thin-walled elastic chests and when practised carefully a very accurate means of outlining the heart. I have repeatedly compared its findings with those of the two other methods, especially plessimetric, and find it satisfactory and trustworthy. One occasionally encounters chests in which for one reason or another it is next to impossible to determine the deep limits of the heart in the ordinary fashion. It is well in such cases to try the method under discussion, since it will often help one out of his dilemma. I should not recommend its employment to the exclusion of the plessimetric method, but merely as an adjunct thereto.

Palpatory Percussion.—By this term is meant a method of using both palpation and percussion at the same time. In other words, it is a method of ascertaining the heart's resistance, and thereby of ascertaining its outline and dimensions. It makes use of the feeling of resistance rather than of the auditory perception of differences in sound. Auenbrugger and Laennec percussed the chest-wall immediately—that is, without the intervention of a plessimeter; the former, by striking with the tip of his finger, and the latter with the end of his stethoscope. It is needless to say this mode of performing percussion is more or less painful to the patient. In 1877 Ebstein proposed palpation of the heart and other solid viscera, as the liver, as a means of appreciating their size by their resistance, and at the International Medical Congress at Rome in 1894 he read an elaborate paper in which he discussed and explained his method at considerable length. In this paper he called attention to a method employed by J. Hein, which consists in palpating the heart with one finger while percussing with the other in the following manner: The palmar surface of the terminal phalanx of the outstretched middle finger is placed upon the chest, while a light tap is made on the chest with the tip of the bent forefinger (Figs. 5 and 6). Then while the extremity of the first finger rests against the wall of the thorax he gives a light blow to the chest with the pad of the middle finger. In each instance the fingers are allowed to remain for an instant in contact with the part percussed, so as the better to perceive the sensation of resistance imparted. In this way, by alternately tapping with the

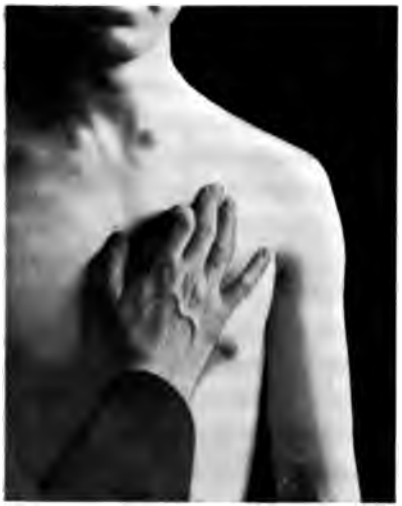


FIG. 5.—HEIN'S PALPATORY PERCUSSION. First position.

two fingers, the entire area is traversed. This is said to yield very accurate results, but is by Ebstein considered inferior to his method, because not altogether devoid of pain to the patient. Ebstein, therefore, makes use of a small glass pleximeter, upon which he gives a gentle pressing stroke with the tip of one finger, which, flexed at its metacarpal articulation, is held slightly and rigidly curved as the stroke is given (Fig. 7). The blow is not made with a quick rebound (staccato), but with a firm pushing movement (legato). The stroke is given in a line perpendicular to the surface thus percussed and the pleximeter is held firmly in position. Ebstein's pleximeter of glass is ½ an inch (1.3 centimetre) in width, 1⅝ inch (4.0 centimetres) in length, and surmounted by a small handle ⅝ of an inch (1.5 centimetre) in height. With such a pleximeter Ebstein asserts the method is not only gratifyingly precise, as he has repeatedly proved on the cadaver by means of needles, but is easily acquired, which is an opinion contrary to that expressed by Vierordt. Moreover, it possesses the additional advantage of enabling the examiner to avail himself of his perception of the sound and pitch of the note produced, as well as of the sense of resistance. In this way two impressions are received simultaneously which serve to control

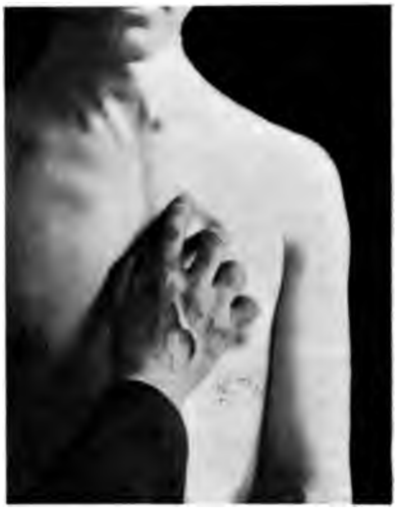


FIG. 6.—HEIN'S PALPATORY PERCUSSION. Second position.

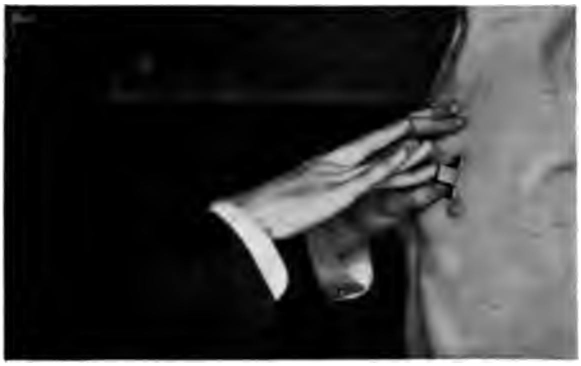


FIG. 7.—EBSTEIN'S PALPATORY PERCUSSION.

each other. Ebstein declares also that by his method one can obtain satisfactory results in cases of emphysema and in persons with a thick panniculus of fat or large mammary glands, all of which usually preclude accurate percussion after the ordinary method.

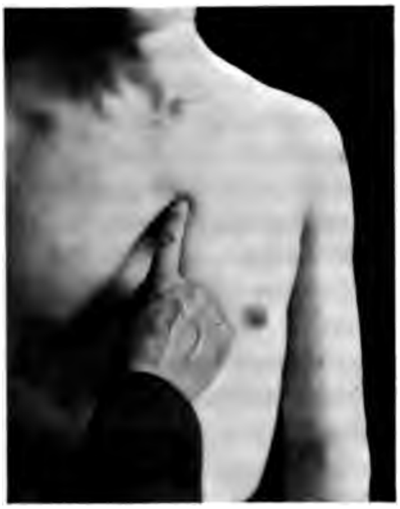


FIG. 8.—MAGUIRE'S METHOD OF PALPATORY PERCUSSION.

Robert Maguire, of England, advocates palpatory percussion by tapping lightly with the soft palmar cushion of the terminal phalanx of one finger, and claims equally accurate results (Fig. 8). He expressly states that the stroke must be not short and quick, but long and pressing, as if one were feeling or palpating with the finger. It is applicable, he says, not only to all solid organs, spleen and kidneys, as well as heart and liver, but also to collections of fluid in thoracic and peritoneal cavities.

In cases which are at all obscure it is well to verify the conclusions derived by any one method—plessimetric, auscultatory, or palpatory—by each of the others. For my part I value the auscultatory method the least highly, because so liable to error in exactly those cases which offer the greatest difficulty to ordinary percussion—that is, emphysematous, fat, and rigid chests.

Auscultation of the Heart is another and indispensable means of making cardiac examinations, and by the inexperienced is apt to be relied upon, if not exclusively, at least to a degree out of proportion to its importance as compared with percussion. Neither can be complete without the other. I desire also to emphasize the folly of attempting to do accurate work without the use of a stethoscope. Whatever form or kind of instrument enables one to hear the most distinctly is, in my opinion, the best for him, regardless of the arguments advanced in favour of certain sorts. I make use of a simple binaural and of a monaural stethoscope, employing the latter when desiring such information as is sometimes obtained from the impulse of the hypertrophied or dilated heart against the chest-wall. A stethoscope with a small end-piece enables one to differentiate sounds and murmurs and to trace them to their source in a way that cannot be done by the ear placed against the præcordia.

Normal Heart-sounds.—The detection of murmurs is not the only object of auscultation. The heart-sounds themselves often afford as much, if indeed not more information than do bruits. Therefore, if one is to correctly interpret what he hears come from the heart, he must be familiar with the characters of the normal sounds of this organ. To this end he must know how they are produced,* and keep in mind what is going on within, during the portions of the cardiac cycle, at the time of the sounds and during the intervals of silence.

If one listens at any point upon the cardiac area he hears two distinct sounds, known as the first and second sound respectively. Over either of the ventricles, in the neighbourhood of the apex, the accent falls on the first, which is longer, of a lower pitch, and more intense—that is, more booming than the second, which is, conversely, short, sharp, and clicking, having a valvular quality we say. Moreover, the ear detects two intervals or periods of silence, of which the shorter occurs during systole between the first and succeeding second sound. The longer, known as the pause, falls between the second and next ensuing first sound, during diastole. This succession of sounds and silences gives to the heart-sounds their peculiar rhythm, likened to the ticking of a clock. If now auscultation be made at the base of the organ, in the second interspace at either side of the sternum, it is perceived that the accent falls on the second sound, since this is the louder and clearer and higher pitched of the two. Their rhythm is, however, the same as at the apex. Furthermore, it is generally perceived that the second sound is louder on one side of the sternum than on the other, the position of greater intensity not always being uniform in different individuals, depending on various conditions, as age, etc.

---

* It is common to speak of sounds, whether normal or abnormal, as produced within the heart or chest. Of course such phraseology is loose and not in accordance with the known laws of acoustics. Sounds are the auditory perception of waves imparted to the air by the vibration of structures within the thorax, the tissues serving as good conductors of these vibrations. With this understanding of the mode of production of these acoustic phenomena, I shall, for the sake of convenience and the avoidance of circumlocution, speak of sounds as generated in the heart or chest.

What occasions this slight diversity between the sounds at the apex and base? Why do not the two sounds in all situations have the same character? I will answer the latter query first. The first sound is synchronous with the apex-beat, and is therefore produced during ventricular systole. Physiology teaches us that the duration of this phase of the cardiac cycle is $\frac{35}{100}$ of a second, subdivided as follows (Fig. 9): During the first tenth of a second the ventricle is initiating its contraction and is silent; during the following $\frac{15}{100}$ of a second its contraction reaches its maximum energy, the auriculo-ventricular valves close, and the first heart-sound is heard; the final tenth of the second, during which the ventricle still remains contracted, is again a period of silence and terminates the phase of ventricular systole.

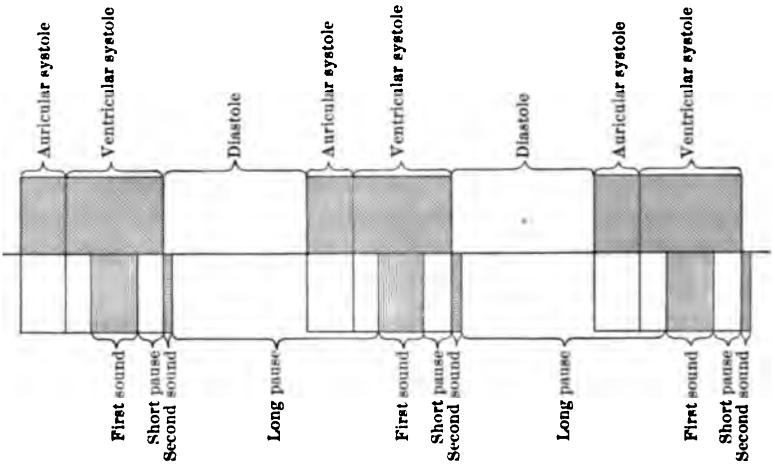


Fig. 9.—Normal Cardiac Cycle.
Phases of cycle above line; sounds below.

During the stage of active contraction blood is being forced from the ventricles into the aorta and pulmonary artery. With the completion of this propulsive stage the ventricles relax; arterial walls recoil, forcing the mass of blood against the sigmoid valves, which, thus thrown into tension and closed, give forth a tone, the second sound, which signals the closure of the valve and the commencement of diastole. This sound is, therefore, diastolic, and, ushering in the stage of cardiac repose, is succeeded by the period of silence or long pause.

This brief statement of what takes place during the different phases of the cardiac cycle will help us to understand the mode of production of the two sounds. During the middle portion of systole, when the first sound is audible, the ventricle is actively contracting and the auriculo-ventricular valves are closed and held closely in contact through the contraction of the papillary muscles. Experiments have shown that if either participant in this stage can be made to act without the other a sound is still audible, but it has lost its normal character. If in the bloodless heart the ventricles are made to contract while the auriculo-ventricular valves are hooked back, the sound is low in pitch, prolonged, and booming, while if the ventricle be opened and the valves closed without contraction of the muscular walls, the sound produced is higher pitched, shorter, and less intense. It is thus apparent that the first cardiac sound is a composite one made up of two elements, a muscular and a valvular.

On the other hand, the second sound is due solely to the vibrations generated in the semilunar valves at the instant of their closure and possesses no muscular element. It is consequently of higher pitch, shorter duration, and less intensity than the first sound. Inasmuch as the first is a composite sound, it is obvious that its two elements must synchronize exactly if the sound is to be pure and normal. Furthermore, there are two ventricles and two sets of auriculo-ventricular valves. Consequently each half of the heart is responsible for its own first sound. Auscultation at the apex, however, reveals but one first sound, which is the result largely of the blending of the two sounds generated in the two halves of the organ, but conducted to this point. That this is the case is proved by the clinical experience that occasionally over one or the other ventricle the systolic sound is heard to be of altered quality or divided into its two elements, while over the opposite half of the organ it retains its normal characters.

Inasmuch as there are two sets of semilunar valves, there are two separately produced yet synchronous *second sounds.* Of these, the aortic is heard most distinctly at the right edge of the sternum in the second interspace, while the area of greatest audibility for the pulmonic is in the corresponding interspace at the left border of the sternum. In the early years of life, by some said to be up to the thirtieth, the pulmonic second sound is the louder of the two, while at and after middle age the reverse obtains.

Conditions which raise blood-pressure in either the lesser or greater system will correspondingly alter the intensity of these sounds. The more feeble first sound heard at the base at either sternal margin is probably transmitted thither from the respective ventricle. Tiegerstedt says it is not impossible that vibrations caused by the opening of the semilunar valves play a certain rôle in the production of the first heart-sound. If this be the case, then the systolic tone audible at the base of the heart in the aortic and pulmonary areas respectively, is not to be regarded merely as a conducted sound transmitted thither with less intensity than to the apex.

Reduplication of the Heart-sounds.—Either the first or second sound may under certain conditions be doubled—that is, divided into two parts or split, as is sometimes said. This occurs most frequently with the second sound, and is best heard over the base of the heart. It may be perceived if the breath is held at the close of a deep inspiration, and under these circumstances is spoken of as physiological. *Pathologically* such a reduplication is apparent when in consequence of disease there is an alteration of blood-pressure in either the pulmonic or aortic system. It is most frequently observed in mitral or pulmonary diseases which augment blood-pressure in the vessels of the lesser circulation. It has been contended that in such a condition the valves at the pulmonic orifice close slightly earlier than do the aortic curtains, and emit their sound an appreciable interval of time in advance. Opponents of this theory admit the lack of synchronism in the closure of the two sets of signoid valves, but maintain that the increase in blood-pressure causes a delay, not a premature occurrence of the sound, since to overcome the unnatural resistance in the pulmonary artery the ventricle is compelled to contract more slowly (Barr). In other words, the ventricle, whether right or left, depending on the system in which blood-pressure is raised, completes its systole perceptibly later than does its fellow. Guttmann's theory of the doubling of the sound being due to asynchronous closure of the individual leaflets of a valve is regarded as fallacious. Reduplication of the second sound, therefore, is an indication of some alteration of blood-pressure in one or the other system.

There is another form of doubling of the second sound which, among English writers, who appear to have paid particular attention to this anomaly of the heart-sounds, is distinguished from the foregoing as *apparent* or *simulated doubling.* This variety, if I may so term it, is heard only at or near the apex, and appears to be confined to cases of mitral disease with predominating stenosis. The phenomenon is believed to be due to the addition or interpolation of a *third* sound closely following the physiological *second.* The only theory regarding its mode of production that appears tenable is, so far as I am able to learn, that advanced by Sansom. He believes it to be a sound of tension in the altered segments of the mitral valve. Upon the occurrence of ventricular diastole the mass of blood held back during systole in the left auricle and pulmonary veins rushes forcibly into the rapidly relaxed ventricle, and, streaming in the direction of least resistance, fills the space behind the thickened and displaced mitral cusps, " bellying them out," after the manner of sails filled by the wind. This sudden bulging of the diseased curtains produces a sound of tension which is audible in the fore part of diastole soon after the normal second sound, which in mitral stenosis, in Sansom's opinion, is the *pulmonic* second, transmitted to the apex. The *aortic* second is, he thinks, too feeble to be heard at the apex. That this third element of sound is produced at the mitral orifice seems supported by the observation that it occasionally becomes transformed into a diastolic murmur occupying the same position in diastole—i. e., following the normal second sound. The diagnostic value of this seeming doubling of the second sound at the apex will be discussed in the chapter on Mitral Stenosis.

Sewall likewise attributes this reduplication of the second sound to the tone of valve-tension, but explains it on the hypothesis that the irritable papillary muscles, stimulated by the inrush of blood from the overdistended auricle, contract too soon—i. e., in the fore part of diastole. This explanation may hold for those cases in which doubling of the second sound is a transient phenomenon, as heard sometimes during states of great cardiac excitement, but not for cases of mitral stenosis.

Reduplication of the First Sound.—Under certain conditions, as that of abnormal blood-pressure within one or the other ventricle, there is heard not a single first sound, but a *reduplication* or *splitting* of this sound. This abnormality is less frequently perceived than is doubling of the second sound, and is equally difficult of satisfactory explanation. Two main theories are advanced to account for it. One of these finds its causation in a hemisystole, by which is meant the separate and independent contraction of the two ventricles. Although there have been recorded a few cases in which highly competent and careful observers believed they detected such hemisystole, still it is so at variance with the physiology of the cardiac action to suppose the ventricles can fail to synchronize in their systoles that many authors are not willing to accept this explanation. The other theory assumes that the two components of the first sound in one or the other side of the heart do not fall together, but are separated by a brief yet distinct interval of time, so that to the ear the first sound over that ventricle gives the impression of splitting or reduplication. One or the other constituent of the sound is generated either too soon or too late to synchronize with the other. As the phenomenon occurs when blood-pressure in one of the ventricles is too high, and as under these conditions the cardiac wall has lost its normal tonicity, it seems reasonable that the tension into which the valves are thrown and the contraction of the heart-wall should not be perfectly simultaneous. Sewall argues that the cause of the reduplication lies in the failure of the papillary muscles to contract at their proper time, their contraction, and hence the note of valve-tension, occurring either before or after that of the ventricular wall. Whatever be the true explanation of this phenomenon, its occurrence betokens excessive, and it may be dangerous, increase of pressure in that ventricle, to which the reduplication can be traced. It may be audible over either half, and I recall a case of mitral regurgitation in which this doubling appeared in the right ventricle only when the patient assumed the recumbent posture. It is not seldom present over the left ventricle in cases of chronic nephritis, and then betokens dangerous excess of blood-pressure in the arterial system, and, secondarily, within the left ventricle.

Gallop or Canter Rhythm.—A phenomenon, sometimes observed and due to the interpolation of a third sound (which, according to its position in the diastole, produces an apparent reduplication of either the first or second sound), resembles so closely the hoof-beats of a galloping horse that it has been termed the canter-rhythm or *bruit de galop.* The merit of having first described it is accorded by the French to Bouillaud; yet to Potain, but to Barié in particular, belongs the credit of having first brought it to the notice of the profession. Fraentzel has also given a most clear and discerning description of the phenomenon based on accurate scientific observation. When the peculiarity under consideration is present, the auscultator hears not merely two sounds of normal relative strength and rhythm, but three, of which the last is an accidental or interpolated sound occurring in the long pause. François-Franck, according to Sewall, is authority for the statement that this third sound may occur in any one of three positions: Immediately after the normal second, in the middle of the diastolic interval, or at the end of the long silence shortly before the first sound. When it falls directly after the normal second sound, it must not be confounded with the apparent doubling of the second sound already described. It is distinguishable from this latter by its peculiar tempo. Its canter rhythm is imparted to it by the shortening up of the long interval and by the accent falling on the middle one of the three sounds—i. e., the normal second (Fraentzel). If one will imitate the sound of a slow canter by striking his hands on his knees, he will at once appreciate the correctness of Fraentzel's statement.

Any one, however, who has studied this rhythm of the heart-sounds in a large number of cases will have appreciated the fact that it not infrequently possesses the characters of a rapid gallop rather than a slow canter. When such is the case, Fraentzel's description does not apply. The tempo and accent are now changed, as may be proved by again imitating the sound by the hands. It will now be observed that the interval separating the first from the second sound is shorter than that separating the second from the third or interpolated sound, while the accent falls most sharply sometimes on the first, sometimes on the third, but in every case least strongly on the middle one of the three sounds. In still other instances the rhythm described by Fraentzel is maintained, but the accent is on the first sound, thus producing a not quite typical canter-rhythm. It is this lack of uniformity in rhythm and accent, which, as it seems to me, explains the diversity of opinion expressed by different writers.

Potain's explanation of the phenomenon is that it is due to an increase in the elastic resistance of the ventricular wall over its muscular tonicity, in consequence of which the inrush of blood from the auricle causes it to generate a sound of tension. Sewall, on the other hand, attributes it to the contraction of the papillary muscles taking place prematurely—i. e., during diastole. Whatever be its mode of production, this rhythm is an evidence of abnormal blood-pressure within the ventricle, and hence of dangerous tension of its wall. Its occurrence is most commonly observed over the left ventricle in cases of chronic nephritis, particularly the interstitial variety, and when thus observed it is to be regarded as an evil prognostic omen. It indicates a giving way of the ventricle, which is no longer able to cope successfully with the resistance in the arterial system.

I agree fully with those who look upon it as a sign of the end being not far distant in cases of chronic nephritis, since I have never known an individual to recover in whom this rhythm was detected. In the spring of 1900 I had under treatment a comparatively young man, with stiffened arteries and interstitial nephritis, who presented this phenomenon at different times in its most typical form. Several times, under the influence of nitroglycerin and cathartics, his gallop-rhythm disappeared entirely, becoming replaced by two heart-sounds of normal rhythm. Yet so soon as pulse-tension was increased, either through lessened vigour of this medication or the administration of digitalis, the ominous disturbance of rhythm reappeared. This patient succumbed after about two months.

This interesting canter-rhythm is never heard at the base of the heart, but always over one or the other ventricle, and consequently in either the mitral or tricuspid area. It may be of transient duration, yet is often persistent. It may be heard in hypertrophy alone or combined with dilatation, it may occur in dilatation alone, in acute infectious diseases, such as typhoid fever and diphtheria, croupous pneumonia, scarlatina, acute articular rheumatism, and acute miliary tuberculosis (Fraentzel), all of which lead to myocarditis or to simple weakness of the heart-walls. And lastly, it may be heard in pernicious anæmia, leucæmia, and grave cachexiæ, which induce profound cardiac asthenia and consequent want of tonicity.

According to French authors, it sometimes occurs over the right ventricle in cases of gastric disease, and Johnson says it may be produced by pulmonary emphysema. Fraentzel mentions it as occurring in other lung affections, leading to dilatation and hypertrophy of this right chamber, with marked cachexia. I once observed a true gallop-rhythm in the fourth and fifth right interspaces close to the sternum, for a brief time, during which there was very obvious overdistention of the right cavities secondary to a rheumatic mitral regurgitation. The very unusual situation of the rhythm in this instance is only explicable by the supposition that in consequence of the enormous distention of the right ventricle the auriculo-ventricular sæptum had become pushed so far towards the right that the wall of the ventricle extended to the fourth and fifth right interspaces. It disappeared so soon as treatment had unloaded the cardiac chambers.

Murmurs.—This is a comprehensive term, which includes all those adventitious acoustic phenomena connected in some way with the heart's action and not resembling in tone the normal cardiac sounds. They may be primarily divided into *endocardial* and *exocardial.* The *endocardial* are subdivided into organic or structural and inorganic or accidental, called also functional, anæmic, hæmic, and dynamic. *Exocardial* are divisible into pericardial, pleuropericardial, and cardio-pulmonary.

By organic murmurs are meant such as owe their origin for the most part to structural defect or alteration of the cardiac orifices or valves—in other words, to definite pathological changes of the structures recognisable after death. *Accidental* murmurs cannot, on the other hand, be ascribed to definite pathological lesions, and therefore have received a variety of appellations in accordance with the various theories offered in explanation of the phenomena.

Endocardial Murmurs of Organic Origin.—These were once thought to be caused by friction of the blood in its passage over the roughened inner surface of the heart. This theory was shown to be untenable as long ago as 1847, when it was replaced by the one now generally accepted—namely, that currents or eddies are produced in the stream of blood, the same as in any other fluid, whenever it passes a point of constriction in its channel or flows suddenly into a portion of the containing-tube, which is wider than that directly above. These eddies and currents in their turn generate vibrations which are audible. These secondary currents are the fluid veins first demonstrated by Savart, but applied by Chauveau to the explanation of vascular and cardiac murmurs.

Some of the conditions governing their production in the vascular system are the following: Constriction of the coats of a vessel by external pressure; projection into its lumen of calcareous plates or masses capable of turning the blood-stream from its direct course; aneurysmal sacs or vascular dilatations into which the blood-stream may swirl; and in the heart itself, all pathological changes by which orifices are narrowed and valves rendered incompetent. In addition, murmurs can be produced by vibration of thin membranes and bands as the blood-current sweeps over them.

In Virchow's Archives, Band cxl, is one of a series of suggestive papers, by Richard Geigel, wherein he takes exception to the prevailing notion concerning the causation of endocardial and vascular bruits. By a series of mathematical formulæ Geigel endeavours to prove that if murmurs of the pitch of those usually heard were produced by vibrations in the blood-stream these would have to be of a length that would be physically impossible within the cardiac cavities. He therefore states that the origin of bruits in eddies and currents is utterly impossible, and declares them due to transverse vibrations of the walls of the structures inclosing the blood-stream. His line of reasoning is ingenious, and to my mind has much to commend it, since the generally accepted theory is not altogether satisfactory.

It is this consideration which makes me venture to dwell for a few moments on the explanation of murmurs offered by Davidson, of Edinburgh. According to his theory, murmurs are due to vibrations set up in the valves by the impact of the blood-stream at an oblique angle. By numerous experiments he claims to have demonstrated that when a stream of fluid was injected into a rubber balloon or a portion of the small intestine, one end of which was tied securely about the nozzle of the syringe while the other was tightly ligatured, the fluid veins and eddies thus generated at the end of the nozzle within the elastic receptacle did not produce more than a very faint murmur, audible by means of a binaural stethoscope. When, however, the fluid was made to strike the inner surface obliquely a distinct clear sound was generated, the intensity of which depended upon the force of impact. By reducing the rapidity and force of the stream Davidson was able to produce murmurs of varying loudness and roughness. By another set of experiments he was able in the same manner to generate an aortic systolic bruit.

The conditions which favour the generation of organic vascular and cardiac murmurs are multiform, and hence such adventitious sounds vary in respect to intensity, pitch, quality, and duration. They also obey the laws of conduction and are propagated in different directions, according to the seat and time of their production. Moreover, two murmurs of independent rhythm may be generated at the same orifice, or two or more may be produced simultaneously at different locations. So that if one is to differentiate endocardial murmurs, and correctly interpret their significance, he must be familiar with these various characteristics.

The *intensity* of a murmur bears a direct ratio to the amplitude of vibrations in the blood-stream, and therefore to the force of cardiac contractions, and *is not at all a criterion of the gravity of a lesion.* The forcible escape of blood through a small fenestration in a valve-segment, in itself a comparatively trifling regurgitation, may be declared by a very loud murmur that is audible to the patient, or even to a bystander a number of feet distant. Thus Miller and Gibbs narrate the instance of a girl who presented a murmur of such intensity that it could be plainly heard 12 feet away when the listener was in the same room and patient fully dressed, and 3 feet distant when separated from the patient by a closed door. On the other hand, a very grave valvular affection may, if cardiac power is feeble, occasion a scarcely audible murmur or even none at all. It is well known, for example, that a presystolic murmur of mitral stenosis, intense while the heart is strong, may fade away to complete inaudibility when the heart becomes feeble.

Conversely, a murmur scarcely audible during a period of cardiac asthenia may grow in intensity as heart-power is regained. This is the case particularly in aortic regurgitation. In the examination of a patient we therefore avail ourselves of the knowledge that forcible cardiac action intensifies a murmur by having him jump about or otherwise excite his heart to bring out an otherwise faint or inaudible murmur.

Posture also influences the loudness of these sounds, some being more plainly, others less distinctly, heard in the recumbent position. Those of stenosis are more intense in the erect posture, while those of regurgitation are so in the recumbent. The reasons for such variations in intensity are based on the influence of the force of gravity, which is greater in some than in other positions (Gibson). Mitral systolic murmurs are nevertheless often louder in the upright than the supine posture, an effect to be attributed to the greater vigour of ventricular contraction when the patient stands. There are so many exceptions to the effect ordinarily exerted by position that a patient should always be examined sitting, standing, and reclining.

The *pitch* depends upon the rapidity of the vibrations producing the murmur. Therefore, some murmurs are low-pitched, while others are high. The union of overtones with the fundamental tone determines *quality*, and as pitch and quality go hand in hand, low-pitched murmurs are apt to be rumbling, growling, rasping, etc., while shrill ones are often musical, whistling, filing, sawing, twanging, and the like.

Finally, the *duration* of murmurs is variable, depending on the length of time the vibrations endure. Other things being equal, it requires more time for the blood-stream to pass through a narrowed orifice than it does for it to regurgitate through wider ostium whose valve is defective, and therefore direct murmurs, as those of stenosis are called, are generally of greater duration than are the indirect ones of valvular insufficiency. It may be stated as a general proposition, therefore, that the murmurs of obstruction are less intense, lower in pitch, less musical in quality, and of longer duration than are those of regurgitation, which, for the sake of emphasis, may be conversely stated to be higher, louder, more musical, and shorter. There are, however, exceptions to this law. Fortunately, murmurs generated synchronously yet at different ostia are never identical in these four characteristics, and hence are usually distinguishable from each other.

It is also of the utmost importance to note the *rhythm* of murmurs, since in this way alone can be determined in what period of the cardiac cycle they are produced. They are either systolic or diastolic. Even the murmur of mitral and tricuspid stenosis is diastolic, since it occurs during the pause; yet, as it is generated at the time of auricular contraction—that is, immediately prior to ventricular systole—it is commonly designated as presystolic, or, as proposed by Gairdner, as auricular systolic.

The *transmission* of a murmur is along the surrounding solid media, and in the general direction in which the stream producing it flows. It is also governed largely by the intensity of the murmur. Fortunately for diagnosis, it is this law of conduction which aids in the tracing of a murmur to its seat of production. As already stated, the anatomical locations of the four orifices with their valves are so closely related within a circumscribed area that if the sounds, of whatever nature, were not propagated to certain regions where they can be heard with maximum intensity, their correct interpretation would be vastly more difficult. Every examiner of experience has realized the truth of this in the not very infrequent cases in which murmurs are widely conducted and yet not most distinct in their own areas.

Cardiac Areas.—These are four in number, corresponding to the ostia, and are definitely located in circumscribed regions on the chest-wall, where the respective valve-sounds and murmurs are heard most clearly (Fig. 10). Thus the *aortic area* is located at the junction of the second right interspace and corresponding costal cartilage with the border of the sternum. The sounds, whether normal or adventitious, which are here the loudest, are generated at the aortic opening. The *pulmonary area* lies in the corresponding situation at the opposite or left edge of the breastbone. The

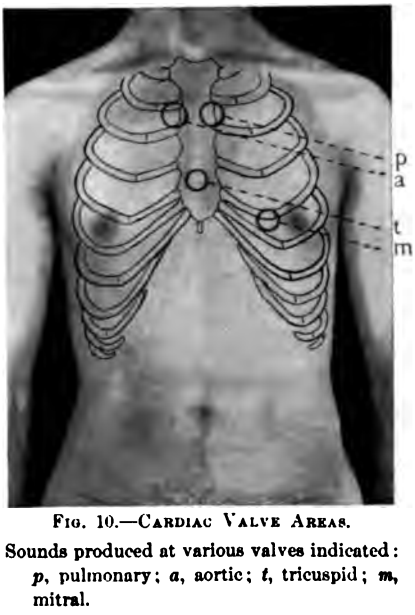


FIG. 10.—CARDIAC VALVE AREAS. Sounds produced at various valves indicated: *p*, pulmonary; *a*, aortic; *t*, tricuspid; *m*, mitral.

pulmonic sounds and murmurs are heard with maximum intensity in this area, although other bruits may be transmitted thither more often perhaps than to the aortic. The *tricuspid area* is located at the lower end of the sternum and corresponds quite accurately to the anatomic seat of the right auriculo-ventricular orifice—i. e., between the fourth left chondro-sternal articulation and the junction of the fifth right costal cartilage with the sternum. Aortic, diastolic, and mitral systolic murmurs are frequently very distinct in this area, while tricuspid bruits may often have their greatest intensity at a short distance therefrom, at either side or below. The *mitral area* is located at the situation of the apex-beat, but is not confined to this. Aortic regurgitant bruits are often transmitted, though feebly, into this region, and mitral systolic murmurs are sometimes even more audible at some point above and to the inner or outer side of the nipple than directly at the apex. Details regarding the conduction of the various murmurs may be found in the respective chapters on valvular affections.

Before leaving the subject of organic murmurs, although still more applicable to accidental ones about to be considered, I wish to caution against the error of relying upon these abnormal sounds in the diagnosis of heart-disease to the exclusion or subordination of other physical signs. In a sense, murmurs are only guide-posts which point out the way one is to look. They are highly valuable signs, but the information they furnish should be confirmed by secondary physical signs, if it is to be taken to indicate valvular disease. A murmur may mislead one because accidental, and the failure to hear a bruit may do the same, but secondary signs will not, because they are founded on changes in the heart and circulation brought about by the valvular defect. The reader will find more on this topic in the section devoted to valvular lesions.

Accidental Murmurs.—These are adventitious sounds heard in cardiac neuroses and certain blood-states, as chlorosis and various forms of anæmia.

Numerous terms are employed to designate this class of murmurs, as functional, inorganic, hæmic, anæmic, spanæmic, and dynamic. The first two imply that there is no structural cardiac affection, and that the murmurs are in some way dependent upon perversion of the heart's function. Hæmic, anæmic, and spanæmic commit one to the proposition of an altered blood-state being responsible for the murmurs. The appellation dynamic carries with it the assumption that the acoustic phenomena depend upon vibrations set up by powerful, perhaps irregular and faulty, action of the heart-muscle. The term accidental sufficiently declares its own meaning, and implies nothing more than that the murmur is a chance result of cardiac action.

Theories to account for these murmurs are many and various, and so long as the condition or conditions governing their production are not definitely ascertained there can be no term that is not open to objection. These abnormal sounds may be heard in any situation over the organ, but are most frequent in the pulmonic and mitral areas. They are systolic and have a blowing or bellows-like character. Such competent and intelligent observers have advanced diverse theories in explanation of these murmurs that it seems to me the part of wisdom to assume that no one hypothesis is applicable to all cases. May they not have their origin in a variety of conditions, some within and some without the heart? I shall describe briefly only the more important theories.

Naunyn explained the systolic murmur heard in the pulmonary area in cases of chlorosis and other depraved blood-states as being in reality due to mitral regurgitation, and assumed that, instead of obeying the law usually governing its propagation, it is conducted along the left auricular appendix to the tip, which, as we have seen, lies directly beneath the chest-wall in front, sometimes overlapping the base of the pulmonary artery. This theory was warmly supported by Balfour, but appears now to meet with general disapproval. Russell proposed two theories, of which one attributed the murmur to narrowing of the pulmonary artery by pressure upon it of the dilated left auricle. In other cases he believed a murmur of tricuspid insufficiency was transmitted into the conus arteriosus, which, in consequence of dilatation of the right ventricle, became displaced outward in the second left interspace. Hanford claims that the phenomenon, which is either heard only or intensified in the dorsal decubitus, results from the pressure upon the artery of a flabby and dilated heart. Foxwell agrees with Russell as regards pressure in some cases of the dilated left auricle upon the artery, but explains other cases as due to a displacement upward of the pulmonary artery and a change in its axis and that of the right ventricle, in consequence of which its normal curve is increased and it is flattened somewhat against the wall of the chest. Bramwell attributes the murmur to the sudden discharge of a large wave of blood of abnormal composition into the probably dilated artery. Sansom thinks that in a condition of right-ventricle weakness toiling to overcome increased resistance in the pulmonic system fibrillar tremors can be initiated at the overstrained portion of the right ventricle—i. e., the conus just below the valves—and in this way the murmur in question can be induced. Gibson holds that auricular or cardiac dilatation cannot be assumed in these cases because the murmur occurs long before such dilatation takes place; also that the experiments on which Foxwell's view is based were faulty; also that if Sansom's theory is correct, then the murmur ought to exist more often than it does, and therefore advocates the view that it is the murmur of tricuspid insufficiency propagated into the pulmonary area. Quincke, cited by Balfour, concluded, as a result of observations in 6 cases of healthy hearts and arteries, but with retraction of the lung-borders, that a systolic basic murmur can be produced by pressure by the heart of the pulmonary artery against the chest-wall.

Vierordt agrees with Sahli that in many cases *venous* murmurs are transmitted from the great intrathoracic veins to the heart. Potain urges the cardio-pulmonary origin of accidental murmurs, maintaining they are generated by the impulse of the heart's apex against the lung, an hypothesis that appears supported by an observation of François-Franck's, who, during an operation upon a dog, detected a systolic murmur in the region of the apex which disappeared so soon as the processus lingualis was lifted away from contact with the heart, and returned when this portion of pulmonary tissue was allowed to again rest against the surface of the organ. Such cardio-pulmonary origin is especially claimed for the murmurs of anæmia. Winckler, on the other hand, believes he has discovered the origin of accidental apex-bruits in a defective action of the papillary muscles or a faulty insertion of the valve-muscles, which permits of regurgitation.

Finally, it has been urged that these murmurs may have a hæmic origin in cases of pernicious and other grave secondary anæmias, while opponents of this view urge the clinical observation that in such blood-states murmurs are not always present, and, on the other hand, occur when anæmia does not exist. Bearing on this objection are the experiments of Thalma, who found that partial exsanguination of dogs did not give rise to accidental murmurs. A condition of overfulness of the vessels caused by the injection of a warm saline solution into the femoral vein was followed by their appearance.

The number and diversity of the foregoing theories serve but to emphasize the sad fact that in medicine there are still many phenomena which have to be accepted as facts, without a satisfactory explanation. In respect to the origin of accidental murmurs, therefore, we can but place ourselves in a judicial attitude and await further proofs.

Musical Murmurs.—These are here introduced because I propose to classify them, not according to their acoustic characters or rhythm, but as organic and accidental, depending upon the anatomical conditions underlying them. First, organic musical murmurs are those not infrequently heard in clearly demonstrable cardiac affections, usually valvular. In their time they may be systolic or diastolic, and in pitch and timbre they are variable. Thus they are described as sawing, filing, buzzing, whistling, etc. Their intensity may be such that the patient is annoyed by the murmur, and it is audible several feet distant, or it may require close attention for its detection. Regurgitant musical murmurs are, as a rule, more intense than direct ones. Yet I recall an elderly gentleman who presented a systolic aortic bruit of a strikingly sawing quality so loud as to be almost painful to the ear. In the case of a negro observed in my dispensary service some years ago there was an aortic diastolic murmur which was audible a short distance from the chest and had aroused the wonder of its possessor. It was not constant, and when present wholly obscured a soft diastolic murmur that was appreciable when the musical one was silent. Each time the sawing sound was present it was accompanied by a thrill in the third left interspace near the sternum of such intensity that it tickled the palm of the palpating hand. This bruit disappeared some weeks prior to death, and at the autopsy no cause for its peculiar quality could be discovered other than the sclerotic and incompetent semilunar valves. In another man, with a bruit of almost identical characters, excepting that it was constant, the necropsy revealed sclerotic aortic valves, one of the cusps being fenestrated, and there being two thin fibrous bands stretched between the edges of two of the curtains. This patient was a pauper at the Cook County Poorhouse, and before the autopsy could be made his body was confided to the tender mercies of one of the medical colleges. It was there found, and the heart secured after a lapse of three weeks. The heart was injured by the preserving fluid, pale and softened, so that during the examination of the delicate fibrous bands they were ruptured. Before the photograph was taken two threads of sewing cotton were passed through the edges of the valves in representation of the bands. The examination and preparation of this heart, shown in Fig. 11, were made by Dr. W. A. Evans. In this instance the fenestration permitted reflux of the blood-stream and the regurgitant wave set the bands to vibrating, and thus occasioned the murmur and accompanying thrill over the body of the heart. Engel has reported a similar case, in which a fibrous band was stretched across the aortic orifice to a pocket of one of the cusps.

The Russian, who under the name of Lewis travels from one medical school to another to exhibit himself to the students, is the proud possessor of a "musical heart." In his case the singing bruit is systolic and of maximum intensity over the right ventricle, and by some observers has been thought to indicate tricuspid insufficiency and to be generated in the right ventricle at the auriculo-ventricular orifice during the reflux.

In addition to fibrous bands or cords, some of the conditions causing a murmur to have a musical quality are said to be vibrations imparted to the thin, stiffened edge of a cusp or fenestration, or to a delicate atheromatous plaque by the blood-stream as it passes over them. In a case of aortic stenosis with a loud systolic musical murmur reported by Mayne, two fibrous bands were found stretched across the cavity of the ventricle just below the greatly narrowed orifice. In another case of mitral insufficiency, which during life had exhibited a musical murmur at the base and a systolic murmur at the apex, Potain discovered post mortem a cord which passed to the wall of the ventricle from the edge of the anterior mitral valve just below the aortic orifice. Demange reported a case of tricuspid regurgitation in which the musical murmur was evidently due to a fibrous band stretched across the interior of the ventricle close to the tricuspid ring. Schroetter has suggested that a musical murmur may be generated by the vibration of a tendinous cord swinging free in the ventricle, or by one that, as a result of endocarditis, had been ruptured and subsequently attached in an abnormal situation. It is needless to remark that the musical quality of these murmurs possesses a pathological interest, but scarcely a diagnostic significance. At the most we cannot do more than conjecture their mode of

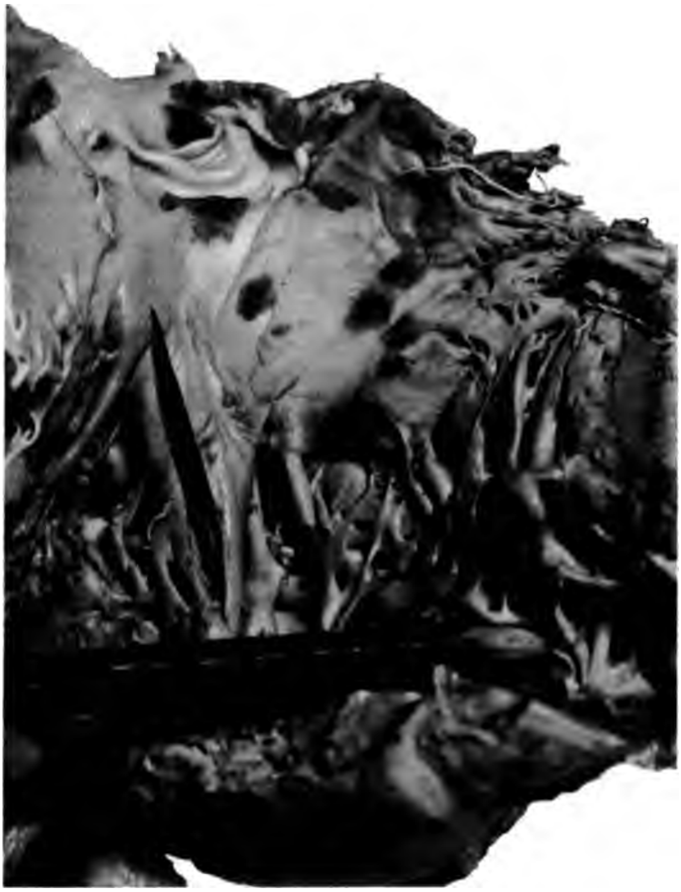


FIG. 11.—INTERIOR OF LEFT VENTRICLE.

Showing fibrous band connecting aortic cusps and responsible for musical murmur.

causation during life until the true condition is revealed by the autopsy.

Accidental musical murmurs are rare, and yet that they do occur is attested by the following case: Miss V. was referred to me by Dr. Charles True, of Kankakee, in the spring of 1897, because of attacks of intense nervousness and agitation accompanied by palpitation and præcordial pain, for which no adequate cause in the heart had been discovered. The patient was a farmer's daughter, nineteen years of age, tall and slender, and gave no history of articular rheumatism or any other infection that would have led to inflammation of the cardiac structures. Family history was also negative. The girl was extremely excitable and unable to give a very lucid or intelligent description of her symptoms further than that she often became frightened, at what was not at all clear, apprehended some imaginary danger to herself or family, and had rapid beating of the heart. During my examination she was much agitated, and the heart action was greatly accelerated, about 120, but perfectly regular. The area of cardiac dulness, both superficial and deep, was not increased, but there was a blowing systolic murmur at the apex, the heart-sounds being sharp and ringing. She was moderately anæmic, and there was a slight enteroptosis. Aside from a not very troublesome fermentative indigestion and constipation, her functions appeared to be normal and the urine was negative. The case was considered one of cardiac neurosis, the murmur accidental, and treatment consisted of hæmatics, laxatives, and remedies designed to lessen the indigestion. The patient was seen by me at rather infrequent intervals, and each time appeared to be somewhat improving. Repeated examinations of the heart failed to elicit anything more than at her first visit, and the murmur subsequently disappeared. On one occasion, however, she seemed more than ordinarily perturbed, and her pulse was more rapid than I had ever seen it. During my examination of the heart, which was always made as a matter of routine, I was astonished to hear over the body of the right ventricle a distinct, short, exquisitely twanging murmur of very high pitch and pleasing quality. It seemed, as well as the tachycardia would allow me to judge, of a systolic rhythm. The action of the heart at the time was extremely rapid and violent. This interesting, and to me exceptional, phenomenon lasted for several minutes, indeed so long as the rapidity of cardiac action endured. When at length her pulse grew more quiet the musical murmur became inaudible and did not reappear. This patient was seen by me in September, 1900, after a lapse of more than a year from her last visit, and although I diligently sought for the twanging sound and any signs of cardiac disease, I failed to detect any abnormality. The patient reported herself as in much better health and less excitable, being but rarely annoyed by her former symptoms, and indeed appeared not the least disturbed by the examination.

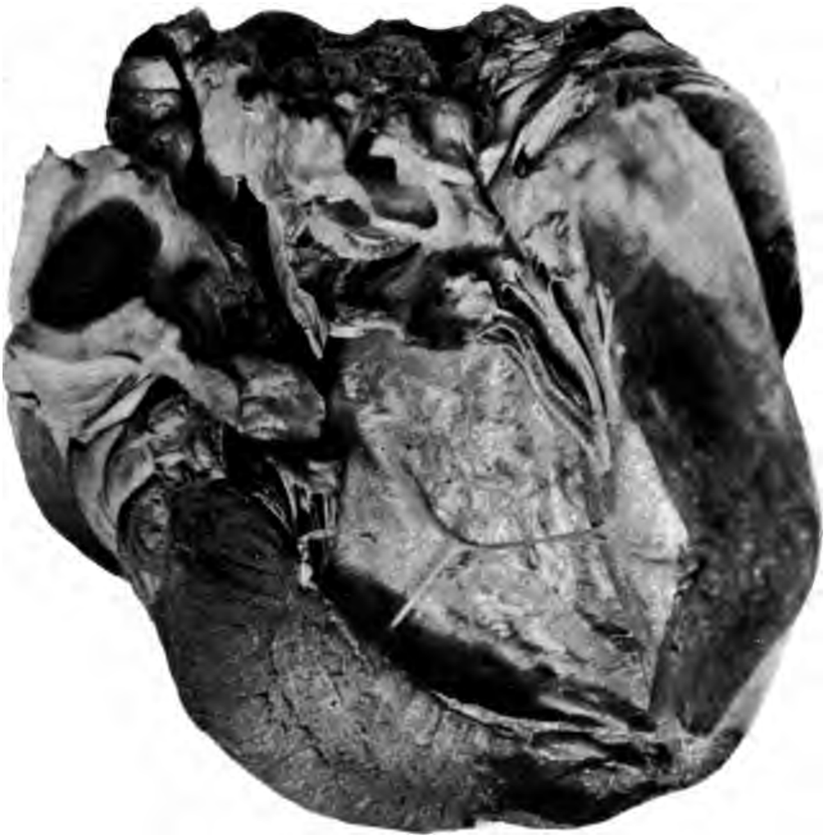


Fig. 12.—Heart of a Buffalo Calf.
Showing aberrant chordæ tendineæ in left ventricle.

The only explanation that has seemed to account for this remarkable phenomenon is that the musical murmur was due to the vibration of one of the so-called *aberrant cords* (Fig. 12) or *mod-*

erator bands, of which so admirable an account has been given by H. F. Lewis. These aberrant cords are thin fibrous bands which, most often discovered in the left ventricle, are seen running along the inner aspect of the wall, or stretched across the upper part of the cavity from one side to the other, or from a papillary muscle to the septum. They have nothing whatever to do with the chordæ tendineæ, and unless sought for are likely to be severed in the opening of the ventricle and thus escape detection. Although without question most frequent in the left, they have yet been found in the right ventricle, and there are rare instances of such a band passing from the valve of the foramen ovale into the cavity of the left ventricle and attached to a leaf of the mitral valve. It is supposed that the function of these moderator bands is to strengthen the cardiac walls in times of overstrain. In the case just narrated it is assumable that in consequence of the tachycardia the cardiac chambers became overfilled and an aberrant cord was thus put on the stretch. In this state of tension it was set to vibrating by the energetic and rapid cardiac contractions, and thus generated the twanging murmur in the same manner as in the case of a violin-string twitched by the finger of the musician. Lewis says it is these aberrant cords which are responsible for the systolic, and sometimes diastolic, musical murmur heard before death. As we have seen, several of the musical murmurs observed in connection with valvular defects have apparently been due to aberrant fibrous bands, so situated as to have been thrown into vibration by the blood-stream.

The Differential Diagnosis of Accidental Heart Murmurs.—Under some circumstances it may be a matter of no small difficulty to differentiate these from organic murmurs. The patient's anamnesis is to be carefully considered, since, if painstaking inquiry fails to elicit a history of articular rheumatism or any infectious disease likely to have set up an endocarditis, it furnishes some evidence in favour of the non-organic nature of the murmur. This is strengthened if the patient is manifestly neurotic, anæmic, or chlorotic, if there are digestive or pelvic disorders that are likely to produce disturbance by way of the sympathetic nervous system, if by reason of the patient's excitability the heart's action is easily perturbed, or if there is a history of cardiac overstrain. If the individual is given to vicious habits, as sexual excesses of one kind or another, particularly masturbation, or indulges too freely in tobacco, tea, or coffee, the presumption is strengthened that the murmur is accidental. Of course, these and numerous other factors that are said to afford *prima facie* evidence of the malady being not organic may exist in a given case with an endocardial murmur of valvular disease. There must, therefore, be made a careful examination of the heart and other viscera.

It may be stated as a general proposition that accidental murmurs are not accompanied by secondary changes in the size of the heart or by circulatory disturbances, such as generally attend and depend upon valvular affections. The discovery of hypertrophy or dilatation of the heart makes strongly for the organic and against the accidental origin of a murmur. Smallness, feebleness, and intermittence of the pulse, cyanosis, dyspnœa of effort, hepatic and other visceral engorgement, etc., are not usual accompaniments of accidental murmurs. Such signs of serious embarrassment of the circulation failing, information may be sought for in the rhythm and other characters of the murmur itself. Accidental bruits of cardiac origin are rarely if ever diastolic, whereas organic ones may occur during any or all phases of the cardiac cycle. Leube has never heard such a diastolic accidental murmur, and doubts its occurrence. Although these murmurs may exist in any portion of the præcordia, they are most frequent over the base, in or near the pulmonary area, and next in frequency at the apex in the mitral region. In transmission they are usually rather limited, and such an apex-bruit is not propagated to the angle of the left scapula.

Organic murmurs, on the contrary, occur with frequency in all areas, and often have a wide extent of audibility besides being propagated in definite directions and to considerable distances. In the matter of intensity, murmurs of both organic and accidental origin are variable. Leube is of the opinion that, as a rule, the latter are the less loud of the two, but the reverse occasionally obtains. The murmur of chlorosis and anæmia, so often spoken of as hæmic, which is heard chiefly in the pulmonic area, is generally intensified in the dorsal decubitus, while Drummond states correctly, I believe, that the "neurotypic" (cardio-muscular) becomes less pronounced after rest in the recumbent posture has slowed the heart. This latter type is most intense during excitement and in the standing position. The same is true of the cardio-pulmonary murmur. These last two diminish or disappear in the lateral decubitus, particularly when the patient lies on his right side. Organic bruits never wholly cease in the recumbent or lateral position. Respiratory movements also affect the intensity of accidental, but not the organic murmurs. The basic chlorotic bruit is loudest at the end of forced *expiration*, and at the close of deep inspiration may cease entirely. A neurotypic (cardio-muscular) murmur is also intensified at the end of forced expiration, and less or absent at the close of a deep inspiratory effort. The cardio-pulmonary or cardio-respiratory murmur, on the other hand, is influenced conversely, being loudest at end of forced *inspiration* and weakest at end of deep *expiration*, or ceases when the patient holds his breath.

The pitch of accidental murmurs is usually higher than that of the organic, yet is rarely musical, and may occasionally be lower. In quality the former is apt to be softer, yet may be harsh, even grating, and the apex-bruit in neurotic individuals is not infrequently vibrant or "whizzing" (Drummond), and may be accompanied by a systolic thrill in the upright position or when the murmur is the loudest during excitement. Finally, the pulmonic second sound is not so accentuated, as a rule, in accidental murmurs as in mitral systolic bruits of organic origin. The reason is obvious; in the former there is not the same likelihood of secondary pulmonary hyperæmia. This lessened intensity of the pulmonary second sound goes hand in hand with the absence of appreciable enlargement of the right ventricle.

In a considerable proportion of cases a definite opinion cannot be expressed at the first sitting, and must be reserved until the patient has become accustomed to an examination or has grown less nervous, or until after the results of treatment have been observed.

Exocardial Murmurs.—The pericardial friction-sounds that come under this head will be found fully considered in the appropriate chapter. Pleuro-pericardial murmurs may result from the friction of the heart on the roughened surface of the pleura, where it comes in contact with the former. Their differentiation from pericardial or endocardial murmurs, which they may at times simulate because of the rhythm, is usually accomplished by having the patient hold his breath, when the friction-sound disappears. A deep inspiration often increases its intensity.

SECTION I

DISEASES OF THE PERICARDIUM

CHAPTER I

ACUTE PERICARDITIS

Morbid Anatomy.—The pericardium is a closed sac lined with serous membrane which surrounds the heart, a visceral layer of the serosa (the epicardium) being reflected over the surface of that organ, and for a short distance along the roots of the great blood-vessels. The parietal layer of endothelium is re-enforced by a strong fibrous lamina, extending from the diaphragm below, to be continuous with the fibrous sheaths of the great vessels above. The pericardial sac usually contains after death a few drachms (10 to 15 cubic centimetres) of a clear straw-coloured fluid.

An inflammatory process of the pericardium may involve only the serosa, or may penetrate into the myocardium or into the fibrous tissue of the parietal layer. It may involve the entire surface or it may confine itself to limited areas, single or multiple, thus giving rise to the circumscribed form of the disease. The morbid anatomical condition is the same in the two forms except in the extent of involvement, and the same description applies to both.

The first evidence of inflammation is the injection of the blood-vessels lying beneath the transparent serous membrane, the process usually beginning in the parts of the sac surrounding the great vessels. This is associated with considerable desquamation of the endothelium, which gives an appearance that is described as of having been breathed upon. As the endothelial cells are those which lubricate the surfaces, this desquamation occasions friction between the two layers of the sac, giving rise to the sounds that are heard during life. This is the simplest form of pericarditis, and the disease may proceed no farther. Usually, however, exudation occurs, and the formation of the exudate is in many cases the prominent feature of the disease. The character of the exudate varies extremely. It may be fibrinous, serous, purulent, or hæmorrhagic, sero-fibrinous, or fibrino-purulent. In fact, almost any combination may occur.

In the *fibrinous, dry*, or *plastic* form the exudate appears at first as a thin smooth pellicle, of a grayish-white or yellowish-white colour, easily detached from the injected surface beneath. Later the exudate becomes thicker, and is of a pasty consistence, and not so easily detached from the underlying surface. The incessant motion of the heart causes the plastic exudate to assume forms that have been variously described by different authors. A common condition is one resembling the appearance produced by tearing apart two pieces of thickly buttered bread. At other times fine threads of fibrin attached all over the surface of the pericardium give to the heart the shaggy or hairy appearance that has received the names of *cor hirsutum, cor villosum*, and *cor tomentosum* (Fig. 13). In still other instances the fibrin is arranged in coarser masses of the characteristic grayish-yellow colour. Such an exudate, being in contact with both layers of the pericardial sac, forms between them adhesions of the kind described as recent or fibrinous, in contradistinction to the old or fibrous adhesions found in the chronic form. The appearances described are to some extent the result of the tearing apart of the two layers of the sac, thus loosely bound together. The processes leading to the repair of this lesion are those eventuating in chronic pericarditis, and are considered in that connection. According to Osler, plastic pericarditis is frequently tuberculous, but the tubercles are very easily overlooked in the presence of the fibrinous exudate.

When the exudate into the cavity is of a fluid nature the condition is known as pericarditis with effusion. The effusion may be serous, purulent, or hæmorrhagic in character, but the most commonly occurring condition is that in which the effusion shows mixed or intermediate characters. In the serous form there is an effusion of serum from the inflamed surface, which may be perfectly clear, but more commonly contains fibrin in the form of shreds, flakes, or larger masses, which may float in the fluid, or

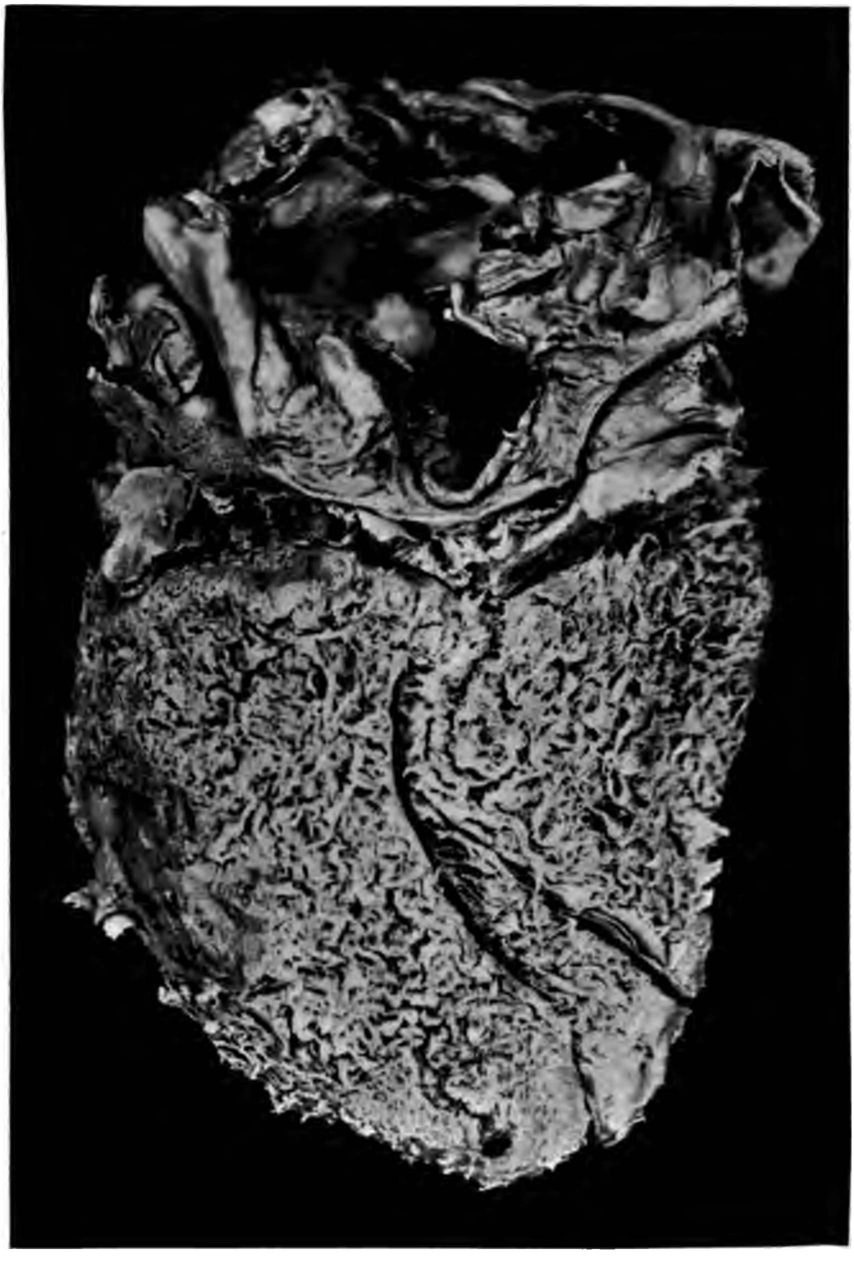


FIG. 13.—COR VILLOSUM OF ACUTE PLASTIC PERICARDITIS.
Photograph of specimen in Museum of Cook County Hospital.

may be deposited on the walls of the sac as a creamy layer. This is the form of the disease known as *sero-fibrinous pericarditis*, and is the one most commonly met with. It usually begins as a dry pericarditis, the effusion developing later, and indeed in the dry form there is aways some transudation of fluid, although its amount is insignificant compared to that of the fibrin. The fluid is often slightly turbid from the presence of leucocytes, but in insufficient number to entitle the effusion to be called purulent, or a small proportion of blood may give to the fluid a reddish or brownish tinge. The amount of fluid varies from a few ounces to several pints.

In the *purulent* form the effusion is rich in cells, and of a thick, creamy consistence, but all degrees of variation exist between this form and that presenting a serous exudate with slight turbidity from the presence of pus-cells, so that a sharp line cannot always be drawn between the two conditions. When the effusion is truly purulent the condition is practically an abscess, and the pus may burrow and rupture externally, as, for instance, in the first right interspace, or in the neck above the clavicle. The condition is a serious one, and shows but little tendency to resorption, and yet the pus may become inspissated and calcified.

In the *hæmorrhagic* form the effusion contains a large proportion of blood, or even, as in scurvy, may seem to be composed of pure blood. In cases of long standing the decomposition of the hæmoglobin gives the fluid a brownish rather than a red colour. Aside from the scorbutic form, hæmorrhagic pericarditis occurs most often associated with tuberculosis of the pericardium and with malignant disease. The effusion is usually very large, and may take place so suddenly as to produce the symptoms of acute secondary anæmia.

Various bacteria have been found in the exudates of acute pericarditis, including the various pyogenic organisms, the diplococcus pneumoniæ, and the bacillus tuberculosis, but it is not always possible to demonstrate bacteria. The presence of organisms causing putrefaction may give to the effusion a foul odour. The Bacillus aerogenes capsulatus may produce gas in the pericardium. This production of gas is probably a post-mortem change (Osler), but according to Coplin (1899) occurs during life.

Secondary changes are found mainly in the myocardium, which may show inflammatory infiltration, or a fatty or albuminous degeneration of its muscle-fibres, leading, after the acute stage has passed, to an insterstitial myocarditis. There may be evidence of recent disease of the endocardium, usually due to the same morbific agency as the pericarditis. Associated disease of the lungs or pleuræ usually bears an etiologic relation to the disease of the pericardium. If there has been high fever, the various parenchymatous organs show cloudy swelling.

Etiology.—If one compare the statements of older authors with those of modern writers concerning the causation of acute pericarditis, the chief difference that will impress him will be found in the change of views regarding the frequency of the primary as opposed to the secondary form of this affection. The term primary was made to include those cases regarded as idiopathic or of spontaneous origin. A better knowledge of pathology and etiology, founded on the results of bacteriological investigation, has taught us the fallacy of a belief in spontaneous development of disease. Authors now restrict primary inflammation of the pericardium to those cases originating in trauma, and include among the secondary all other cases once considered primary or idiopathic. This is undoubtedly due in part to a more accurate knowledge, and therefore more frequent recognition, of the rheumatic nature of many disorders whose pathology was formerly but indistinctly understood—as, for instance, certain rheumatic nodules occurring in childhood. The chief reason, however, is to be found in the remarkable additions made to our knowledge during the past twenty years or so regarding the pathology and bacteriology of disease, above referred to. At present the physician would be far behind the times who failed to recognise inflammation of the pericardium as a local manifestation of a general constitutional disease or as a secondary infection in the course of some disease having pathogenic organisms as an etiological factor. For instance, the primary pericarditis that was formerly thought to follow exposure or chill was probably due to a rheumatic attack, the true nature of which escaped recognition.

Furthermore, some of the cases of pericarditis, formerly regarded as idiopathic, were observed in individuals whose general resistance had been greatly reduced by privation or chronic alcoholism. In such the pericardial inflammation was, properly speaking, due to infection. The sero-fibrinous pericarditis arising in the course of articular rheumatism is a local expression of the rheumatism, and the suppurative pericarditis sometimes seen in puerperal septicæmia is due to the primary infection. Roberts goes so far as to say: "In my own experience I have never met with an instance of acute pericarditis which, when carefully investigated, could not be included as a secondary event in one or other of the etiological groups now to be discussed." Among these, he includes pericarditis from extension or irritation, from trauma and perforation, from cardiac and aortic disease, and those associated with new growths, general miscellaneous diseases, and blood-states.

In his investigations regarding "terminal infections" in chronic disease, Flexner has made some highly interesting and important observations with reference to the etiology and bacteriology of acute pericarditis. I cannot do better than to reproduce one of his tables, which gives the frequency with which certain bacteria were found and their point of entrance:

Bacteria.	Frequency	Infection Atrium.
Micrococcus lanceolatus	11	Pneumonia, 8 times.
Streptococcus	4	Bronchitis, 2 times.
Staphylococcus aureus	1	Erysipelas, 1 time.
Bacillus pyocyaneus	1	Leg ulcer, 1 time.
Micrococcus lanceolatus and Bacillus coli	1	Peritonæum, 1 time.
Bacillus influenzæ	1	Tonsils, 1 time.
Streptococcus, Staphylococcus aureus, and Bacillus coli	1	Cancer, stomach, 1 time.
Staphylococcus and Bacillus coli	2	Sloughing myoma, 1 time.
Unidentified bacilli	1	Doubtful.

Tubercle bacilli should be added to this list. From the foregoing it is plain that acute pericarditis may be a secondary infection following a great variety of local infectious processes, or it may arise in the course of an infectious disease, and be due to the pathogenic organism of that disease.

Rheumatism.—All observers agree in placing articular rheumatism first in the list of those affections which give rise to acute pericarditis. The certainty of this connection was established by Pitcairn in 1788, although his views were first widely published in 1795. Writers have been in accord concerning their causative connection, yet there has been great diversity of opinion regarding the frequency with which pericarditis occurs in the course of rheumatism; whether it occurs most frequently in the first or subsequent attacks; whether it is most likely to be associated with inflammation of one or several joints, or any particular joint; whether with chronic as well as acute articular inflammation; and whether or not it precedes or follows or develops coincidently with the joint affection. Concerning the first of these questions, it is generally held that pericarditis occurs less frequently than endocarditis, yet there is a wide divergence in the figures given by authors regarding its numerical relation to attacks of rheumatism. Chambers gave it as occurring in 13 per cent, Ormerod in 71.7 per cent, Bamberger in 30 per cent, while Bauer, although believing exact figures cannot be stated, considered from 16 to 20 per cent not far from the truth. Of Poynton's 150 fatal cases of rheumatic heart disease in children, he found evidence of pericarditis in 113 cases (75 per cent). Personally, I regard such statistics as of but small value, and consider it sufficient to state in general terms that rheumatism is so frequently complicated by acute pericarditis that in every case of the former affection the medical attendant should keep a sharp lookout for the development of pericardial inflammation. Most authors agree, I think, in the opinion that pericarditis is more apt to occur in the first and endocarditis in the subsequent attacks of articular rheumatism. Bauer asserts that it bears *no definite relation to the number of joints affected nor to the involvement of any particular joint.* It certainly does not occur more frequently in rheumatism of the upper than of the lower extremities. The last-named author states emphatically, also, that it does *not* occur in the course of chronic rheumatism, nor when but a single joint is affected. The development of acute pericarditis is by others thought more likely in the severe forms of the rheumatic affection, and therefore when a number of joints become attacked.

Although such a relationship between acute pericarditis and severe rheumatism was noticed by Sibson, it was not constant. In children rheumatic manifestations are often mild, and yet the little ones do not escape pericardial inflammation. In one case coming under my notice the pericarditis followed no other evidences of rheumatism than vague pains in the knees, with erythema accompanied by mild fever, these symptoms having been preceded by follicular tonsillitis.

There are no constant time relations, moreover, between an attack of rheumatism and inflammation of the pericardium. The latter may even precede the former, although it most commonly develops during or after the rheumatism. It generally makes its appearance from the fourth to the sixth day of the rheumatic disorder, sometimes not before the tenth or fifteenth day, and has even been known by Sibson to be postponed as long as the sixty-third day. Rheumatic pericarditis may exceptionally attack individuals of all ages, but is undeniably most frequent in young adults who have been rendered susceptible to it by hard work or exposure. In England it appears to be particularly prevalent among young servant-girls below the age of twenty-one (Sibson), and among persons of both sexes thus afflicted at a later age, the majority were found by the same author to follow more or less laborious outdoor occupations.

Its prevalence among children is shown by statistics gathered from children's hospitals by Sturges and Poynton. Yet Roberts states that, according to his experience, pericarditis is very much less frequent in children of the better classes, a fact which, he believes, shows the predisposing influence of hardship, not alone in the production of rheumatism, but also in the development of pericarditis.

Satisfactory evidence of the infectious nature of the rheumatic poison has not yet been adduced, although many observers have expressed the belief that the pathogenic organism will yet be discovered. If such an organism should one day be identified, then pericarditis would no longer be considered a complication, but a natural though not a necessary part of the pathological process of inflammatory rheumatism.

Nephritis.—The importance attached to renal disease in the production of acute pericarditis is scarcely appreciated, I think, by the majority of physicians. A few writers of wide clinical experience place nephritis as only second in this regard to inflammatory rheumatism. It should, however, yield place to acute pneumonia in this regard. The pericardial inflammation is not limited to acute nephritis, as might be supposed, from the fact that the latter is so frequently observed in the course of acute infectious diseases, but may appear during the progress of any one of the chronic forms of kidney disease. Indeed, it is said to be a specially frequent complication of the small red kidney. Uræmia seems to particularly predispose to acute pericarditis, while the supervention of the latter contributes largely to the fatal termination of the primary affection. Most authors content themselves with a statement of the fact and make no attempt to explain the well-known etiological connection between acute or chronic inflammation of the kidney and inflammation of the pericardium. Two explanations may be given, however. By some the blood of nephritic patients is thought to contain some noxious substance, possibly of chemical nature, possibly of catabolic origin, which results from renal disease, and which in consequence of renal inadequacy is not excreted.* This noxa is an irritant, and gaining access to the pericardial cavity, there sets up an irritative inflammation. Givadinovitch expresses the opinion that acute pericarditis in Bright's disease is of true toxic nature. It is mostly fibrinous, but may be hæmorrhagic and very rarely sero-fibrinous, and always occurs in an advanced stage of the renal disease. According to the other less conservative explanation, pericarditis is a true secondary infection, caused by the conveyance to the pericardium of germs circulating in the blood, and responsible for the acute or chronic pericarditis. In cases of the small red kidney, it is assumed that invasion of the pericardium by bacteria takes place either because the renal disease has impaired the germicidal action of the blood, or because it interferes with the proper elimination of the micro-organisms.

Apropos of the statement that infection frequently occurs in Bright's disease, Flexner's observations may again be quoted. Of 32 cases of chronic nephritis occurring alone, in which there was *general* infection, micro-organisms were positively identified in

---

* Chatin has reported four cases of pericarditis in patients suffering from nephritis. In three cases with effusion bacteriologic examination showed the fluid to be sterile. In these three cases the serum was hypertoxic. The toxic elements supposed to be responsible for the inflammation of the pericardium have been found neither in the circulating blood nor in the effusion; and the existence of aseptic and amicrobic pericarditis in certain cases of Bright's disease is well established. The pericarditis of nephritis may sometimes develop as a complication of an ordinary infection, and is usually aseptic or sterile.—Revue de médecine, July 10, 1900.

29. It is worthy of note, however, that in none of these cases was pericarditis present. On the other hand, pericarditis was found 23 times in cases of chronic nephritis in which there was *local* infection, whether the nephritis existed alone or in combination with some other chronic disease, as of the heart or liver. It would seem, therefore, that although a *general* infection may occur in the course of chronic nephritis, pericarditis does not take place unless there be some other *local* infection. In the majority of cases of pericarditis in the course of chronic nephritis there was pneumonia, either croupous or lobular. It may be queried, therefore, whether the pericardial inflammation is not secondary to the local infection rather than to the nephritis itself.

Acute Pneumonia.—This infection should certainly be given a place only subordinate to articular rheumatism in the etiology of acute pericarditis. The frequency of this association has been recognised by authors, but has been brought out with special clearness by Preble, who found pericarditis in 92.4 per cent of 79 cases of fatal pneumonia collected from the post-mortem records of Cook County Hospital. Preble came to the conclusion that the danger of pericarditis bears a direct relation to the extent of lung involvement, and is also relatively more frequent in left-sided than right-sided pneumonias. The inflammation of the pericardium may result from direct extension through the lymphatics or may occur independently, and is due to the pneumococcus, which has been frequently identified in the exudate.

Scarlatina.—This is sometimes complicated by the occurrence of acute pericarditis, and in some cases this has taken place during the stage of desquamation. As the scarlatinal organism has not been identified in the pericardial effusions, this latter is probably to be regarded as a mixed infection due to streptococci or staphylococci. Bauer observed a post-scarlatinal pericarditis coincident with rheumatic manifestations, and was therefore inclined to attribute it to the affection of the joints; but inasmuch as pus germs are often responsible for the rheumatic affection, the pericarditis, as well as the rheumatism in that case, may very well have been an instance of mixed infection following the scarlatina.

Other Infections.—Other diseases in the course of which acute pericarditis has occasionally been observed are erysipelas, smallpox, typhoid fever, measles, cholera, and even diphtheria. It must also be remembered that Flexner found as foci of infection bronchitis, leg ulcer, sloughing myoma, cancer of the stomach, and even tonsillitis and disease of the peritonæum. In some it was probably a secondary event, in others a true mixed infection. When pericarditis complicates acute pleuritis it is generally stated to be by extension. It is, in fact, either a secondary event due to the one and the same cause, or it is a mixed infection.

Acute inflammation of the pericardium has been associated with varying diseases of neighbouring parts—e. g., enlarged glands or tumours in the mediastinum, abscess, or caries of a rib, and has resulted from a rupture into the sac of an empyema, from perforation from an ulcer of the œsophagus or stomach, and even from intraperitoneal abscess. When caused by such conditions the pericarditis is usually purulent. One very remarkable case has been narrated of perforation and inflammation of the pericardium by a set of false teeth which had been accidentally swallowed and had lodged in the œsophagus, where it caused ulceration.

Acute pericarditis is sometimes occasioned by aneurysm of the aorta and by new growths in the pericardial sac—e. g., tubercles. These are capable of setting up an acute inflammatory process of the pericardium, but as a rule the inflammation is subacute or chronic, which probably explains why it so frequently escapes clinical observation. It is doubtful whether gummata ever induce acute pericarditis.

Hæmorrhagic pericarditis occurs as a secondary infection in the course of scurvy, purpura hæmorrhagica, and hæmophilia. Some writers also assert that cancer and tuberculosis induce the hæmorrhagic variety. Ebstein has reported two cases of hæmorrhagic pericarditis, and stated that pericarditis was specially likely to be hæmorrhagic in the chronic or recurring form, and also in the aged and in the hæmorrhagic diathesis. In this condition, he thinks, there is a toxic or infectious cause that creates a tendency to hæmorrhagic exudates. Such changes are at least as important as the mechanical ones. The pericarditis secondary to scorbutus may be regarded as a type of this class. It may also occur in alcoholism, which induces the hæmorrhagic diathesis. In most cases of traumatic pericarditis the blood found in the sac comes from the bleeding wound. Cases of traumatic origin in which the pericardium is not perforated are harder to understand.

Valvular Defects.—Chronic valvular disease seems undoubtedly to predispose to pericardial inflammation; this is said to be particularly the case with aortic insufficiency. Why valvular lesions should thus tend to the production of pericarditis is a matter for conjecture. By the advocates of the doctrine of the infectious origin of all inflammations, it would probably be explained as an instance of secondary or mixed infection, in consequence of the very close anatomical and physiological connection existing between the endocardium and pericardium.

Trauma.—Finally, acute pericarditis is sometimes the result of direct injury, as gunshot or stab wounds, blows upon the chest-wall and laceration by fractured ribs. Under such circumstances micro-organisms are usually introduced into the pericardium, and there set up an acute inflammatory process which, if the cocci be pyogenic, will prove to be suppurative.

DRY PERICARDITIS

SYN.: *Fibrinous, Plastic, Adhesive Pericarditis*

The pathology and etiology of this form have already been considered, and therefore I shall pass at once to

Symptoms.—This disease usually arises during the course of some already existing infectious process, and therefore its invasion, and even its subsequent progress, are likely to be masked for a time by the clinical phenomena of the primary affection. Indeed, some authors go so far as to state that there are so few subjective symptoms attending dry pericarditis that it may be said to be a latent affection. In many instances this is probably correct, but I believe the existence or absence of subjective phenomena is determined by the degree of intensity and extent of the pericardial inflammation.

If in the course of acute articular rheumatism there is a sudden elevation of temperature which cannot be explained by the fresh involvement of other joints, or if delirium or pronounced disturbance of the nervous system suddenly takes place, especially in children, it is suspicious of some of the heart-structures having become invaded by the inflammatory process. This organ, therefore, should at once be carefully examined, and if necessary repeatedly examined, for, according to the figures already quoted from Poynton, the pericardium in children is a specially frequent seat of inflammation. If, as thought by Roberts, the opinion appears to be quite prevalent among general practitioners that acute fibrinous pericarditis is not very frequent among children, and not apt to leave serious consequences behind, it certainly would seem to be in place to again call attention to Poynton's figures. Out of 150 fatal cases of rheumatic heart-disease in children, there was evidence of more or less acute plastic pericarditis in all but 9. In 113 the pericardium was more or less adherent, while in 77 the adhesion was complete. Moreover, the pericarditis appeared to contribute more to the fatal issue than did the endocarditis, for the reason that the inflammatory process extended from the pericardium to the myocardium and led to dangerous dilatation.

Pain.—This is an early and fairly constant symptom, although in some cases it appears to be more like a vague sense of distress than actual pain. It is generally felt in the cardiac region, but may be located in the epigastrium, while in some cases it radiates over the front or side of the chest, even along the course of the brachial plexus into the arm. In a case of this kind described by Sibson there was also endocarditis. Occasionally it is experienced between the shoulders, and is then held to indicate inflammation of the posterior portion of the sac. Bäumler has described pain and sensitiveness on the side of the larynx. In some instances there is associated with the pain such a hyperæsthesia of the skin of the præcordia as to make percussion of the heart almost impossible. Painful deglutition has been frequently reported, and is not difficult to understand when we remember that the pericardium is attached to the œsophagus and would be pressed upon by the ingesta in their passage down the gullet. Patients have also been known to complain of the heart hurting them with each contraction, and it may well be that when the covering of the heart is inflamed pain can be felt every time the organ changes in form during systole.

In character and severity this symptom differs much in different cases. It may be sharp and cutting or dull and heavy. In a case observed recently the patient was only able to describe his pain as a steady dull ache over the heart. Usually the anguish is continuous, although in some cases it is intermittent, coming and going like a veritable neuralgia. In others again it assumes a paroxysmal character. The countenance generally betrays suffering by an expression of pain or distress, and the patient not infrequently keeps his hand upon his heart. Although this symptom, pain, is doubtless due, in large part at least, to the friction produced by the rubbing together of the inflamed pericardial surfaces, still its intensity depends also upon the sensitiveness of the patient, it being well known that some persons never feel pain so acutely as do others of a less phlegmatic temperament. The pain of pericarditis persists so long as the inflamed surfaces continue to rub against each other, and hence when these become separated by effused fluid this symptom abates or disappears. Therefore, if pain suddenly ceases while the continuance of pyrexia points to continuance of the active inflammation, it may be taken to indicate beginning effusion into the sac.

Cough may or may not be present, but when present is usually dry and frequent, and when conjoined with pain may give rise to the suspicion of pleurisy. In a fourteen-year-old girl seen not long ago and in whom the inflamed pericardium had led to great cardiac dilatation, with consequent pressure on the left lung, the attending physician at first mistook the case for one of pneumonia. This case is so instructive that I will briefly report its salient features. On a certain Friday this girl complained of slight pain and stiffness of one of her legs, but was not prevented thereby from going to school as usual. The following Monday she felt several slight chills, which were attributed to the coldness of the room in which she was at the time. For several days following she showed signs of malaise, and in other respects did not seem well, yet did not give up and go to bed. Friday night, a whole week from her initial rheumatic attack, she spent at a friend's house, but when the next morning came was unmistakably ill, and the family doctor was sent for. He found her with a dry cough, hurried respirations, rapid pulse, considerable fever, and a sharp pain in the left side above the heart. Examining the lungs, and discovering some dulness and bronchial breathing at the left posterior base, he pronounced the case pneumonia—an error that could have been avoided by a proper examination of the heart. Three days later another physician saw the patient, and at once recognised the true character of the disease. When on the ensuing afternoon I was called in consultation, the cardiac dulness presented the characteristic triangular outline and a systolic apex-murmur was audible, but the friction-sound had disappeared. The case was one of acute pericarditis, as shown a few days subsequently by the results of aspiration. The amount of effusion was small, however, and the marked increase in the area of cardiac dulness was due chiefly to the dilatation the heart had undergone. It was impossible to say whether the mitral systolic murmur indicated a valvular lesion or was relative in consequence of the dilatation. But as there was a history of some sort of illness three years before, at which time she had "heart trouble," it was feared that the valves were defective and were perhaps sharing in the present inflammation. In this case the pericarditis had probably begun almost a week before she was obliged to give up, so that it is not strange that the process should have induced signs of pressure by the end of the first week. This patient ultimately made a good recovery.

The *pulse* in these cases is accelerated, running sometimes as high as 130, or even 140 to the minute, and is usualy compressible and regular in the early stage before the myocardium has become much affected.

The *respirations* are usually rapid and often shallow, either because the patient shrinks from taking a deep breath, lest the pain be intensified, or because an actual sense of dyspnœa is experienced.

Temperature.—An elevation of body-temperature probably attends most cases of acute pericarditis, but is often masked or modified by the fever due to the primary affection. As a rule, the degree of pyrexia is not great, averaging perhaps 102° to 103° F., and being generally continuous or mildly remittent. When it occurs in the course of chronic nephritis, or when it is associated with chronic myocardial or endocardial lesions and independent of rheumatism, the pericarditis frequently runs its course without fever, or at all events with so slight a pyrexia as to be overlooked. The duration of the temperature is somewhat variable, depending on the intensity of the infection, but may be said to average two to three weeks.

Loss of appetite and other derangements of the digestive tract, as flatulence and constipation, are usually present, the same as in other febrile and acute infectious processes, while the *urine* is scanty and high-coloured. If it contains albumin, this is due to an associated nephritis or depends either upon a primary affection or upon a long-standing visceral engorgement resulting from antecedent cardiac disease and is not due to the pericarditis itself.

Sleep is disturbed or prevented altogether by the pain and nervousness caused by the inflammation. Children are often fretful and restless. The countenance is pale and anxious or expressive of suffering. In the spring of 1901 I treated a gentleman of fifty-five for symptoms of failing heart, the result of chronic myocarditis and associated vascular and renal changes. He was taking a course of Nauheim baths and seemed to be getting on very well, when I left town for a few days. Upon my return I received word that he was very ill and in much pain. I found Mr. H. sitting in a chair looking pale and drawn, and when he spoke it was with a hollow, feeble tone of voice. This was Thursday afternoon. He stated that on the Tuesday morning preceding he had been seized with a dull, heavy pain over the heart, which had not left him for a moment since. He had not slept for two nights, and could not lie down on account of his great shortness of breath. The pulse was 106, weak, inclined to be thready, yet regular. His breathing was not noticeably disturbed so long as he was quiet, but his temperature in the mouth was 101.2° F. Suspecting pericarditis, I yet purposely reserved my investigation of the heart for the last and went over the lungs carefully, finding nothing more than râles of hypostatic congestion at the posterior bases. Coming to the heart I could detect no change over what had been discovered at my last visit, the Saturday previous, excepting that the tones were much more feeble. The area of dulness did not appear increased.

I was about to give up, in doubt of the nature of the trouble, when I chanced to catch in a circumscribed location over the roots of the great vessels at the left of the sternum a soft brushing murmur that had not been there at any of my examinations before. This murmur was systolic and short, not at all like a pericardial rub in rhythm, but upon pressing firmly with the stethoscope I discovered that the murmur entirely disappeared. This convinced me that the case was one of acute pericarditis, and, knowing the feebleness of the degenerated heart, I believed the attack would prove speedily fatal. A mustard-plaster, followed by hot fomentations, was ordered, and a nurse was at once secured. At my next visit, four hours later, the pain was mitigated somewhat, but the patient's condition was manifestly worse. Strychnine, $\frac{1}{30}$, was ordered hypodermically every two hours, and in addition $\frac{1}{8}$ of morphine with atropine was injected. I left him, feeling that the night was to prove a critical one, and at midnight I received a telephone message that Mr. H. was failing rapidly, his breathing being very laboured, and his pulse at the wrist too rapid and thready to be counted. A physician living close by the patient was sent at once and began the administration of stimulants, but with no apparent effect, as the patient died two hours later. It was subsequently stated to me that as his condition grew worse the pain became less. Consciousness was retained to the last. No autopsy was held, but I believe that effusion began to take place, which relieved the pain by separating the inflamed surfaces, and at the same time overpowered by its pressure the degenerated myocardium, which led to rapid asystolism.

The insidiousness of onset yet intensity of subsequent symptoms are well shown by the case of Mrs. B., a Norwegian, aged twenty-eight, who consulted me in April, 1887, "for heart trouble." Her mother had died of rheumatic heart-disease under my care, and her younger sister had mitral regurgitation, also of rheumatic origin. Six years previously, after the birth of her only child, the patient had articular rheumatism and was ailing for a year, yet had not had symptoms of heart-disease afterward. In December, 1886, she had rheumatism in right knee, both elbows, and left shoulder. Three weeks before coming to me she had begun to suffer from præcordial pains, dyspnœa, and palpitation, each heart-beat accompanied by pain, which was increased by deep breathing and lying down.

Percussion occasioned pain, the pain being most marked over the sternum and adjacent left intercostal spaces, from the second to the sixth, particularly in the third and fourth. The patient's face was dusky, the eyes dull, and a systolic pulsation was visible and palpable in the pulmonic area. There was slight epigastric pulsation, and the pulse was regular and feeble. The apex-beat was in the fifth left interspace, somewhat too far to the left, quick, and accompanied by a feeble thrill. Cardiac dulness was increased in all directions, and in the mitral area there was a loud, harsh systolic murmur transmitted to the back. All the sounds, especially the pulmonic second, were sharply accentuated, and over the base of the heart was a triple murmur that by its rhythm and other characters was plainly a pericardial friction-rub.

Excepting retraction of their anterior margins the lungs were negative. Her temperature and urine were normal. The diagnosis was mitral insufficiency of rheumatic origin, and acute pericarditis, probably plastic, and also rheumatic.

Patient was sent home to bed and a blister was applied to the præcordium. At first, after rest in bed, local applications and salicylate of soda, the patient's condition improved, and she was allowed to get up at the end of ten days. In a few days, however, she again took to her bed, and from this time forward her symptoms steadily grew worse. Cough became very troublesome, with difficult mucous or muco-sanguineous expectoration, and there were anorexia and constipation. The pulse always remained at 120, and as it failed to be slowed by digitalis, the drug was discontinued. June 2d there was a sudden attack of acute rheumatism in the left hand and wrist with substernal pain, and temperature rose to 102° F. Salicylate of soda gave prompt relief to pain, and as the urine was scanty and acid, the salicylate was discontinued for the bicarbonate of potash, which was administered until the urine became alkaline. June 6th, at 2 A. M., there was a sudden exacerbation of substernal pain and distress. A pericardial friction-sound now developed over the body of the right ventricle, chiefly below and to the left of the ensiform appendix. There was great epigastric tenderness and interscapular pain. The anterior margin of the left lung became somewhat more retracted, and the apex-beat now moved nearer to the left anterior axillary line. The patient complained much of pain across the front of the chest, along the lines of the diaphragm, from the right inframamillary to the left infra-axillary region. She complained bitterly of pain in the pit of the stomach, and suffered with nausea and vomiting. June 8th found patient much distressed for breath and unable to retain food. Epigastric pain diminished, but condition of the heart very much as before. Fever was 102° F. at 8 P. M. Stimulants and food in small amounts were ordered. At 11 P. M. there were sudden defervescence, and profuse perspiration for the rest of the night. June 9th patient orthopnœic, pulse 138, unequal, and weak; pain abated; but patient restless. Examination revealed dulness of left base, as high as lower angle of scapula. Expectoration scanty; cough almost impossible; passed a very bad night; opiates given freely. June 10th, summoned hastily at noon to see patient. Abdomen very distended with gas; breath very short; heart very feeble; carminatives, stimulants, and enemata ordered, but very little relief obtained. Death at 7.30 P. M. Treatment throughout tonic, supporting, sedative, and antirheumatic.

Autopsy by Dr. Elbert Wing nineteen hours after death. The inner surface of the pericardium was covered here and there with loose fibrous threads, which presented the appearance of *cor villosum,* while upon both the anterior and posterior surfaces of the heart was an area of recent pericarditis. The sac contained a small amount of serous effusion. The myocardium showed changes of chronic myocarditis, probably dating from the time of the previous attack of pericarditis. The mitral valves gave evidence of chronic endocarditis that had led to their insufficiency, and showed also the effects of recent endocarditis. There was acute circumscribed pleuritis of the left side with about 8 ounces of sero-fibrinous effusion. In the right pleural cavity were old pleuritic adhesions. The lungs were hyperæmic and œdematous. There was subacute diaphragmatic peritonitis, also subacute splenitis, and passive congestion of the liver. Kidneys and other organs were negative.

In some patients, particularly children suffering from acute articular rheumatism, there may be marked symptoms pointing to profound disturbance of the nervous system. These are jactitations, subsultus tendinum, cerebral excitement and restlessness, and low muttering delirium.

It must not be supposed that all the foregoing symptoms are of a necessity present in any one case of acute fibrinous pericarditis, or that they always have the gravity just described. In one patient pain is the chief complaint, another may be annoyed by persistent palpitations, others may manifest no particular disturbance either of the heart or nervous system. Unless the pericarditis is associated with inflammation of the endocardium, dyspnœa is not likely to be marked until the acute inflammatory process gives place to extensive effusion. Respiration may be accelerated, but there is not actual air-hunger.

In many instances, as previously stated, this affection remains so latent that if the physican were to rely for its detection upon subjective manifestations, the disease would surely be overlooked. For this reason the medical attendant should make daily examinations of the heart as a matter of routine practice, in all cases of rheumatic fever or other infectious diseases capable of lighting up pericardial inflammation.

Course and Termination.—If an acute dry pericarditis is circumscribed, the plastic exudate not involving the whole sac, the activity of the process may speedily subside, and all evidence of its existence disappear in the course of a few days or a week. If, on the other hand, the inflammation is intense, and involves the myocardium, or if the plastic exudate is poured out over the entire organ, the course of the disease may extend over several weeks. In such cases, particularly in children with already existing valvular disease, death is not unlikely, or if the patient recovers, he is likely to be left with a damaged heart.

Acute cardiac dilatation is not infrequent, as shown by Poynton's statistics. Indeed, all clinical observers of much experience with pericarditis in children have come to look upon dilatation of the heart as a quite general result, and to regard its occurrence with considerable apprehension. The extension of the inflammation to the myocardium is a matter of grave danger, and one that is likely to result fatally. If fibrin be deposited in a thick layer over the entire surface of the dilated organ, it may act as a mechanical hindrance to the subsequent return of the heart to normal size. This extensive fibrinous exudation results, furthermore, in an adherent pericardium, which will be described in a subsequent chapter.

Physical Signs.—*Inspection.*—From the very nature of acute fibrinous pericarditis it is evident that no information of more than a merely negative kind can be derived from an ocular examination of the patient. The countenance may express anxiety or suffering, and inspection of the chest may note some disturbance of respiration or an exaggerated and rapid heart-beat; but if there be evidence of deranged circulation this will probably be found due to associated cardiac disease, as acute endocarditis, myocarditis, cardiac dilatation, or a chronic valvular defect.

Palpation.—In some cases the hand, or, as preferred by Roberts, the tips of the fingers, laid gently on the præcordium, detects a vibration or fremitus, which is the tactile impression produced by those conditions that give rise to the pericardial friction-sounds subsequently to be described. If felt at all, this fremitus is detected over the body of the heart, usually in the second or third intercostal space, not far from the left sternal margin. It may, however, in rare instances be detected at different points throughout the præcordium. Unfortunately this sign is not often present, but when it exists, it conveys the impression of a rubbing or grating of two rough surfaces, a sort of " to-and-fro " or back-and-forth rub, which is not strictly synchronous with cardiac systole and diastole. It is this peculiar gliding character of the friction-fremitus which readily enables one to distinguish it from an endocardial thrill. Pressure may modify the intensity of this fremitus: moderate pressure increasing, forcible pressure diminishing or obliterating it altogether.

Percussion.—In this form of pericardial inflammation the outline of cardiac dulness may only be affected in so far as this disease leads to dilatation of the heart; in other words, percussion reveals nothing characteristic of plastic pericarditis, or that will be of material service in arriving at a diagnosis.

Auscultation.—In the early stage of acute pericarditis of whatever form, and it may be throughout the entire course of dry pericarditis, auscultation furnishes for the most part our only means of diagnosis. Normally, the two pericardial surfaces glide over each other without friction and noiselessly. But when one or both of them have become roughened by fibrinous exudation more or less friction of movement is occasioned, and this is declared by the so-called pericardial friction-sound.

Before describing this in detail, it may be well to state that a pericardial friction-sound has also been detected independently of pericarditis. It may be produced by the milk-spots usually found on the inferior surface of the right ventricle, also by concretions (Bauer); by dryness of the serous surfaces (Collin and Walsh); and by viscosity of the pericardium during an attack of cholera (Pleischl). Nevertheless, such facts do not vitiate the truth of the statement that in the recognition of the pericardial friction-sound lies our best and usually our only reliable means of arriving at a diagnosis.

Location of the Pericardial Friction-murmur.—As this exocardial murmur, as it is called in contradistinction to endocardial murmurs of valvular disease, is often very circumscribed, it is important to know where it is most frequently and best heard. This is generally over the body of the heart at the origin of the great arteries upon which the pericardium is reflected, or in some cases upon the anterior surface of the right ventricle, very rarely at the apex of the heart. Consequently this friction-sound is audible at the left of the sternum in the second, third, and fourth left intercostal spaces in the same locality as that in which friction-fremitus is commonly felt (Fig. 14). In some instances of extensive pericarditis it is heard at scattered points or throughout the præcordium.

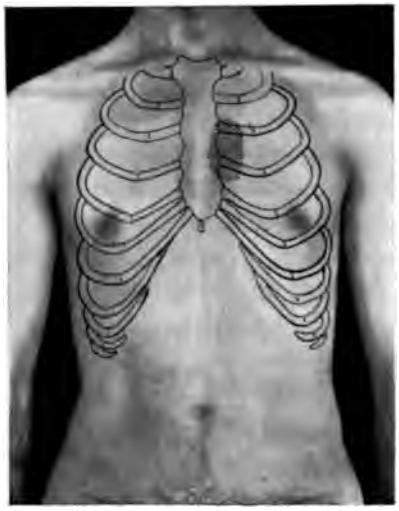


FIG. 14.—USUAL LOCATION OF PERICARDIAL FRICTION SOUND AND FREMITUS.

Rhythm of the Friction-sound.—This is the most important feature of the pericarditic rub, and the one upon which dependence is chiefly placed in the interpretation of its nature. It is very variable, but whatever its peculiarity in any given case, it is as a rule not limited to systole and diastole, as are endocardial murmurs. Instead of being synchronous with either the first or second heart-sound, or bearing a definite relation to these tones, the pericardial rub seems to overlap them or to occur at a time that is wholly independent of them. Thus, according to Skoda, it may accompany, precede, or follow the heart-sounds in what seems to be a sort of hit-or-miss fashion. The rhythm is very difficult to describe, but when once heard in a typical case is again easily recognised. In most instances the friction-murmur is composed of either two or three parts, and when of but two, has a to-and-fro or back-and-forth rhythm, after the manner of a double aortic bruit, but distinguishable from this by its time and quality. The variability in the rhythm of this sound is owing to the fact that the roughened pericardial surfaces are made to rub against each other either during contraction or relaxation of the auricles or during the corresponding phases of the ventricles. Therefore, when this friction-murmur is made up of three parts, one is presystolic, produced by the systole of the auricles, and the other two, of longer duration, fall in the systole and diastole of the ventricles. Very infrequently, according to Bauer, each side of the heart can produce a systolic and a diastolic rub of different duration, so that each heart-beat may be accompanied by four murmurs. Very rarely also a friction-murmur is synchronous with either one or the other heart-sounds, and when this is the case its duration is greater than that of the tone it accompanies, a circumstance by which its true character can generally be recognised. If in such a case one is in doubt as to whether the murmur is exocardial or endocardial, he can generally ascertain its nature by noting the effect of pressure, since this exerts little if any influence upon valvular murmurs. Finally, it should be remembered that a friction-sound may disappear for hours together and then again become audible.

Intensity of the Friction-sound.—This depends upon two conditions: (1) the nature of the exudate, (2) the force of cardiac contractions. If the deposit is fresh and semifluid and the cardiac action feeble, the sound of the rub is likely to be indistinct. If, on the other hand, the fibrin is dry and uneven and the heart is beating forcibly, the friction-sound is likely to be loud.

Quality of the Friction-sound.—This differs in different cases, depending probably upon the dryness and viscosity of the fibrin. It may be grating, creaking like leather, crackling like parchment or like the crunching of dry snow beneath the heel, etc., but in my experience is most often of a soft brushing quality, very dissimilar to the timbre of valvular bruits.

The Effect of Pressure on the Pericardial Murmur.—It is usually found that pressure with the stethoscope modifies this friction-sound in its intensity if not its quality. Gentle pressure by bringing the roughened surfaces closer together intensifies it, while firm pressure diminishes or obliterates it entirely. It is sometimes found also that the intensity of the murmur is affected in one way or another by the patient's position, being louder in the erect, weaker in the recumbent posture or the reverse. In some cases also the intensity is affected by respiration, being louder when by forced inspiration the pericardial layers are brought into firmer apposition, and contrariwise enfeebled when separated by expiration. The reverse of this has been observed, however.

There is nothing in acute pericarditis *per se* to cause abnormal alteration of the heart-sounds. As stated by Roberts, either tone may be obscured by an unusually loud and harsh friction-murmur, but in general they are heard through the murmur in those cases in which they happen to be synchronous. When the inflammatory process has invaded the myocardium or has weakened it through dangerous dilatation, the cardiac sounds are likely to become feeble, and the first at the apex may be more or less toneless, but there is nothing in this peculiar to pericarditis. Stasis in the pulmonary system is evinced among other things by undue accentuation of the pulmonic second tone, while in consequence of the feeble discharging power of the left ventricle the aortic second sound becomes enfeebled.

Diagnosis.—The diagnosis of dry pericarditis is not as a rule attended with insuperable difficulty. In cases in which it is latent or its symptoms are masked by those of the primary affection it may be easily overlooked. In most instances its presence is declared by the history of an antecedent or associated rheumatism, by præcordial pain, etc., and by the characteristic rubbing thrill and murmur. When the anamnesis and symptomatology are negative, reliance must be placed upon the auscultatory phenomena, and these failing, a correct diagnosis is hardly possible.

Differential Diagnosis. — This concerns acute endocarditis, pleurisy, and pneumonia. The diagnosis of acute endocarditis is hardly possible unless valvular murmurs and other definite changes in the sounds and shape of the heart and embolic phenomena are detected. The differentiation of endocardial from exocardial murmurs is based on the laws concerning the latter just described, and as a rule is not particularly difficult if due attention is paid to their rhythm.

In acute pleurisy reliance must be placed upon the detection of the characteristic pleuritic rub, and the possible development of pleuritic effusion, since the history and symptoms of a left-sided pleuritis may be very like those of pericarditis. A point of prime importance is that the pleuritic rub ceases when the breath is held, while that of pericarditis does not.

In pneumonia there are the initial chill, the higher continuous fever, painful difficult cough, tenacious rusty sputum, loss of normal pulse-respiration ratio, dulness of one or more lobes, crepitant râles, and bronchial breathing, and, lastly, the termination by crisis after five to seven days.

One would scarcely think that *aortic aneurysm* would be mistaken for pericarditis, and yet I recall two instances in which such was the case. A middle-aged gentleman once consulted me because of pain in the upper front chest. The only abnormal sign was a faint scratching sound in the region of the great vessels not synchronous with either heart-sound. In the absence of other findings, I pronounced in favour of localized pericarditis, and yet four months later I discovered in the same situation a well-marked aneurysm. The second instance was that of a man in Cook County Hospital who presented a to-and-fro rubbing murmur over the base of the heart, also not synchronizing with either cardiac tone, no dulness, no pressure-symptoms, and the necropsy disclosed three small aneurysmal sacs surrounding the base of the aorta. They were of about the size of English walnuts, and the swish of the blood as it entered and left the sacs had evidently occasioned the pseudo-pericardial rub.

Prognosis.—This is always grave, but depends upon the severity and duration of the attack. In children with articular rheumatism an acute attack of pericarditis, even without effusion, is so likely to set up dangerous dilatation of all the cardiac cavities that if the disease is protracted there is imminent danger of a fatal issue. Dangerous weakness on the part of the myocardium is shown by feebleness and muffling of the first sound at the apex, diminution of the aortic second sound, and by a thready and intermittent pulse. Great derangement on the part of the nervous system is also a sign of danger, even though the life of the patient be not immediately threatened. The remote prognosis is unfavourable, since acute plastic pericarditis may be followed by effects that will greatly hamper the heart in the future. Firm adhesions at different points may unite the two layers of the pericardium, which, if they do not become stretched, may ultimately lead to weakness and dilatation of the right ventricle (Broadbent), or the sac may be bound down to the heart throughout, forming what is known as *synechia pericardii*, the baneful effects of which will be described in a subsequent chapter. Broadbent has related a case in which the œdema and other signs of persistent venous engorgement throughout the body were found due to fibrous bands which had partly constricted the right auricle and led to total obliteration of the inferior vena cava.

Although it is stated that the plastic exudate may sometimes be absorbed, this is a very remote contingency, and should never be reckoned upon as at all likely. When the disease is complicated with endocarditis, pleurisy, or pneumonia, or when it occurs in the course of chronic Bright's disease, the prognosis is usually more unfavourable than when it occurs independently or in the course of rheumatic fever.

The mortality of fibrinous pericarditis is not generally considered very great, and yet a study of the 150 fatal cases of rheumatic heart-disease collected by Poynton shows the erroneousness of this opinion. In 34 of his cases myocarditis was present as secondary to pericarditis and death seemed due to the effect upon the myocardium. Even when the inflammation does not extend to the heart-muscle the heart of a child is very likely to undergo a serious degree of dilatation, and when both these conditions are combined with endocarditis recovery is very improbable. This was well shown in the case of the ten-year-old coloured boy, from whom was obtained the specimen shown in Fig. 13. When seen for the first and only time a few days prior to death, this boy was sitting up in bed on account of difficulty of respiration and of pain in the heart-region. His illness had begun with rheumatism and lasted ten weeks, and he had become strikingly emaciated and his countenance showed marks of patient suffering. The thorax and abdomen were distended from just below the clavicles to the umbilicus, were unnaturally broad across the loins, and thus filled out presented a striking contrast to the thinness and smallness of the neck and extremities. Breathing was extremely rapid and shallow, and as evinced by the pulse the heart's action was also rapid and feeble. The skin was dry and scaly and felt hot, although as a matter of fact there was but slight fever.

The cardiac impulse was very feeble, and the apex-beat could not be clearly defined. Absolute dulness was enormously increased in all diameters, reaching as high as the second costal cartilages, and transversely from at least 2 inches to the right of the sternum far beyond the left nipple almost to the anterior axillary line (Fig. 15). This gave to the dulness a pyramidal shape closely resembling the outline of the pericardium distended with fluid, but differing from it in the circumstance that the left border of dulness did not pass outside the limits of cardiac impulse. The heart-sounds were feeble, and all over the præcordium was a loud, harsh systolic murmur, having its greatest intensity in the mitral area and audible throughout the back of the chest. No pericardial friction-rub could be distinguished, but there was one sound that at first was quite misleading.

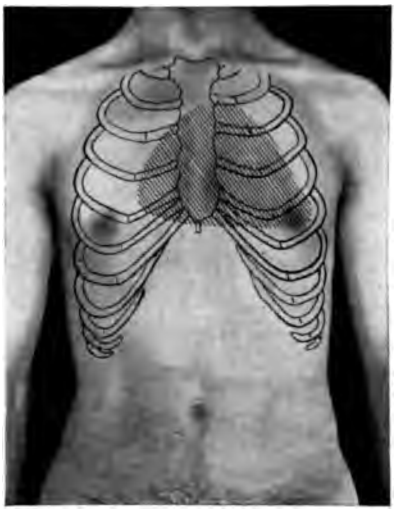


FIG. 15.—ABSOLUTE DULNESS IN CASE OF ACUTE PERICARDITIS.

Beneath the right clavicle, and therefore in proximity to the aortic area, was a double blowing sound, having a to-and-fro rhythm of a quality very like a harsh double endocardial murmur. It was so loud as to obscure the heart-tones, yet, although very rapid, not fast enough to be generated in the heart, and moreover was audible over the back. So soon as these differential points had been noted it was concluded to be respiratory, and accordingly was found to cease so soon as the little patient held his breath. The lung-margins in front were retracted by the pressure of the large heart; pulmonary resonance was impaired to right and left of the heart, as well as at the posterior base of the left lung. There was manifest engorgement of the liver and other abdominal viscera, but there was no œdema.

In the matter of diagnosis it was not so easy to determine whether effusion was present or whether the enormous area of dulness was due to dilatation, as it might at first seem to be. However, by carefully comparing the left lateral limit of dulness with the feeble cardiac impulse and finding that they pretty closely agreed, it was concluded that the condition was mainly dilatation, secondary to acute pericarditis, and probably also endocarditis, with perhaps a small amount of effusion, but certainly not enough to warrant tapping in the hope of relieving the child's dyspnœa. The hopelessness of the prognosis in such a state of affairs was justified by the fatal issue about a week subsequently.

The autopsy disclosed acute plastic pericarditis, without effusion, acute endocarditis, and fatty degeneration of the myocardium, and death was probably to be attributed to the state of the heart-muscle.

PERICARDITIS WITH EFFUSION

Sero-fibrinous.—As already stated, a sharp dividing line between fibrinous and sero-fibrinous pericarditis cannot always be drawn pathologically, because, although a pericarditis may remain dry throughout its course, the fibrinous exudate is generally united with an effusion of serum, so that a process which was plastic at first may afterward be characterized by an effusion of a large quantity of serum within the pericardial sac. The two elements of fibrin and serum may be mixed in varying proportions; in one case the former being abundant, while in another the fluid may contain but an insignificant proportion of plastic material. The amount of effusion in any given case varies within wide limits; there may be 1 or 2 ounces, or the sac may be enormously distended by 1, 2, or more pints (Fig. 16). The effusion generally takes place gradually, but in some instances occurs with such rapidity that the sac becomes entirely filled in twenty-four hours from the onset of the affection.

Purulent.—In this variety the effused fluid is composed chiefly of pus with but little fibrin, and contains pyogenic bacteria. In rare instances the micro-organisms may be of such a nature that the purulent fluid becomes fœtid, and the disease assumes a very grave aspect from the onset.

Hæmorrhagic.—This form is characterized by the effusion of blood into the pericardial sac as a result of the intensity of the process, which undermines the integrity of the pericardial blood-vessels. Or a sero-fibrinous effusion may become deeply blood-stained through hæmorrhages from minute vessels.

These three varieties of effusion may be looked upon as different manifestations of one and the same process, having the same pathology, and differing only in the etiology and intensity of the inflammation.

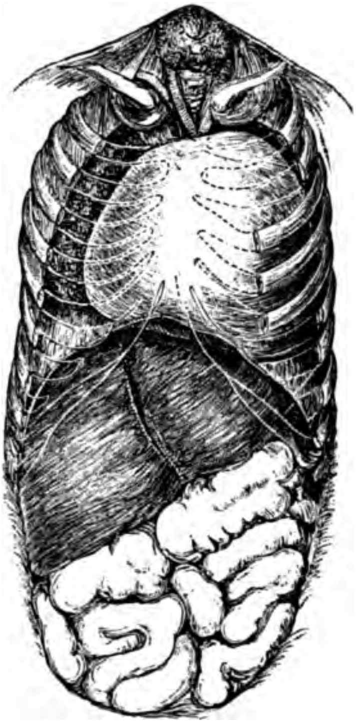


FIG. 16.—CASE OF PERICARDITIS IN WHICH THE SAC CONTAINED 3½ POUNDS OF FLUID. (Bramwell.)

Symptoms.—These are to be divided, according to the stage of the process, into (1) those that attend the onset, and which are chiefly inflammatory in their nature; and (2) those that result from mechanical distention of the sac, which are, therefore, symptoms of pressure on the heart and neighbouring viscera. Furthermore, the three kinds of effusion should be theoretically distinguished one from the other by the severity of their symptoms—that is, of their constitutional effects; as a matter of fact, however, there is often nothing in the symptomatology that declares the nature of the exudate.

The phenomena attending the early or inflammatory stage have been described under Dry Pericarditis, and therefore we may pass at once to the consideration of the symptoms due to fluid accumulation in the sac.

As effusion takes place it gradually distends the sac from below upward, and, separating the roughened and inflamed pericardial surfaces, causes a cessation of the pain attending the onset of the affection. The fever of the inflammatory stage still persists, however, as may also the cough. With distention of the sac, the symptoms due to active inflammation gradually merge into and are subordinated to those occasioned by pressure. When the amount of exudation is small, symptoms of active inflammation may still predominate, but when it completely fills the sac, reaching, it may be, one or more pints, pressure-effects assert themselves, and may even become dangerous.

Children impress me as complaining less of these effects than do adults, yet, of course, individual peculiarity largely determines the amount of complaint upon the part of the patient. I have seen children with an enormously distended sac who yet uttered no word of complaint and whose silent suffering was truly pathetic. They are usually restless, however, and display fretfulness when disturbed. In many cases their patient fortitude as regards subjective symptoms presents striking contrast to the objective evidence of circulatory and respiratory embarrassment.

The face is pale and anxious, or there is congestion of the cutaneous vessels, producing a blue-white appearance, and the veins of the neck are turgid. The *pulse* is small, rapid, and of low tension, which gives it a degree of abruptness that may make it somewhat resemble the sudden pulse of aortic incompetence. Some writers describe the pulse in the stage of effusion as larger and fuller than would be expected from the feebleness of the heart-sounds. Marked irregularity, and even intermittence, are sometimes observed, particularly after the effusion has persisted for a considerable time. Such arrhythmia coming on late is a sign of danger, since it points probably to failure of the heart-muscle. It should not be forgotten, however, that irregularity and intermittence of the pulse may be present from the beginning of the pericarditis, when this latter is associated with a valvular defect, in which case it is not to be attributed to the pericardial effusion or inflammation.

The disturbance of the circulation everywhere evinced is a direct result of pressure on the heart by the abundant effusion. Not only does the heart have to sustain the weight of the superimposed fluid, but when the effusion is great enough to distend the sac, it is confined under high tension and forced, therefore, to press inward on the heart. According to Sibson, the thick-walled and powerful ventricles are better able to withstand such pressure than are the thin-walled auricles and veins, which consequently have their capacity diminished. There is actual mechanical impediment to the inflow of blood into the right heart, and likewise to passage of the stream out of the pulmonary veins into the left auricle. Thus are produced the smallness and weakness of the radial pulse with fulness of the systemic veins.

This is not all of the pressure-effects, however. The distended pericardial sac takes up more room than it did prior to the effusion, and consequently it exerts pressure on the adjacent viscera. It pushes aside the elastic lung-borders; and as the heart lies more to the left than to the right of the median line, it is the left lung that feels the greater pressure. The lower lobe, therefore, is shoved backward to make room for the distended pericardium, consequently the patient suffers from respiratory embarrassment more or less pronounced. Not only are the *respirations* accelerated and shallow, but the patient is compelled to sit up in bed to breathe (orthopnœa), or in some instances to lean forward with his elbows on his knees, so as to allow of as much gravitation of the sac away from the lungs as is possible under the circumstances. No doubt that carbonic-acid intoxication resulting from the mechanical impediment to respiration also plays a part in the production of dyspnœa.

Insomnia is often a very troublesome symptom, and seems to be due not only to passive cerebral congestion, but also to the patient's dyspnœa, which renders it impossible for him to lie down, or speedily arouses him when so fortunate as to fall into even an uneasy sleep.

There is usually *anorexia*; not only has the patient no appetite, but the dyspnœa and *dysphagia* render the taking of food difficult, and children often turn away from it when proffered.

The *urine* is scanty, the abdomen is distended both from retention of gas in the bowels and from congestion in the portal system. The liver is turgid in consequence of mechanical interference with circulation through the lungs, and is more or less tender; there may be constipation or small frequent watery stools, because of serous transudation into the intestines from the engorged vessels within their walls. I have always looked upon this diarrhœa as Nature's effort to unload the distended vessels, and therefore as a very valuable therapeutic hint. If the disease be protracted, and venous congestion very marked, there may even be some œdema of the lower extremities.

Fever is of variable intensity and character; it is likely to abate somewhat as the active inflammatory stage passes into that of effusion, and if this latter stage persists for some weeks the temperature usually returns to normal, or nearly so.

The symptoms of *pressure* as above described usually manifest themselves gradually, but appear suddeny in those cases in which the sac becomes rapidly filled. The gravity of the symptoms usually bears a direct relation to the amount of effusion. When this is small, but 2 or 3 ounces, or when it takes place insidiously in the course of cachectic diseases, symptoms may be entirely latent.

When the pericardial effusion is *purulent*, the gravity of the symptoms depends both upon the amount of the exudation and the kind of micro-organisms concerned in the process. It is in this form that the sac often reaches its greatest degree of distention, and since the degree of mechanical interference with both circulation and respiration accords with the amount of effusion it requires no further comment.

Scarcely had the foregoing been penned when I was asked to see 2 cases of acute rheumatic pericarditis in children, which illustrated so well certain features of similarity, and yet of contrast, that I have decided to narrate them here. A girl of twelve years, seen with Dr. F. S. Johnson, gave the history of a severe attack of inflammatory rheumatism five years previously involving several joints, but the heart was said not to have been affected. She afterward had two mild rheumatic attacks, of which the last was a year ago. During the past summer and fall the patient was thought by her parents to have been remarkably well. About four weeks ago she had an attack characterized by mild fever, coated tongue, and slight jaundice, but no distinct rheumatic symptoms. Ten days ago she was allowed to attend the opera and eat freely of candy, after which the symptoms of two weeks earlier returned with greater intensity.

When Dr. Johnson assumed charge of her case he found the patient with mild intermittent pyrexia, ankles and knees painful, but not red or puffy, slight præcordial pain, great nervousness, restlessness, and so much cutaneous hyperæsthesia, as well as pain, that a very thorough examination of the chest was not possible.

He found weak, rapid, but regular pulse, 120 to 130, and respirations of 60; great increase of both absolute and relative cardiac dulness, particularly upward and to the left; a loud systolic murmur throughout præcordium, but most intense at apex, together with a short presystolic murmur limited to a small area within and above the apex-beat.

The apex-beat was in fifth space outside left nipple, heart-sounds were everywhere audible, pulmonic second banging and split. A pericardial friction-sound existed at the base, over right auricle. Left lung was compressed and the liver palpable just above the level of the umbilicus. There was no dropsy, and the urine was negative.

The case was regarded as one of acute rheumatic pericarditis supervening upon a combined mitral lesion, and having led to great general dilatation of the heart. Three days later temperature was 102° F., pulse 120 to 130, but regular, and respirations 60 to 80. Patient was in evident distress, complaining of pain in the heart above the left nipple. She also had great difficulty in swallowing. A $\frac{1}{12}$ grain of morphine gave her a fairly comfortable night, and the morning when I saw her the condition was as follows: Patient lay nearly flat in bed, several joints of both lower and upper extremities anointed with a liniment containing oil of wintergreen and swathed in bandages. She was excitable, fretful, and inclined to cry out when touched. There was no cyanosis, but respirations were shallow and rapid, 60 or 80 to the minute, the pulse of fair volume, but dicrotic, was about 120, perfectly regular. The abdomen was distended and tympanitic, tense and painful in the region of the liver, which could be made out extending nearly to the level of the umbilicus. The heart's apex was visible and palpable, though rather weak and diffused, in the fifth left interspace outside the nipple-line. There was no pericardial fremitus, but the cardiac impulse was diffused from apex to base and from left mamillary line to the sternum.

Cardiac dulness, both superficial and deep, was increased transversely, but chiefly to the left, the deep limit reaching nearly to the anterior axillary line, but not extending outside of or below the palpable impulse of the apex. A harsh mitral systolic murmur was everywhere audible, as were also both sounds, the pulmonic second being greatly intensified and split. Over the body of the sternum pressure with the stethoscope brought out a soft rubbing murmur, which, from its quality and rhythm, was easily recognisable as pericardial. The inferior boundary of cardiac dulness was not depressed; indeed the abdominal distention occasioned an elevation of the heart.

This high position of the liver caused the upper margin of hepatic dulness to reach the level of the fifth right costal cartilage and interfered with the determination of the presence or absence of Rotch's sign. It seemed to me, however, that the outer border of the right auricle lacked its natural curve downward and inward, and that the line of dulness joined that of the liver at nearly a right angle. The pain which change of position caused the little patient, rendered examination of the back of the chest inadvisable. The doctor stated, however, that the day before he had found dulness with corresponding alteration in the breath-sounds at the left posterior base.

The diagnosis was acute rheumatic pericarditis with great cardiac dilatation and possible acute myocarditis supervening upon a previously existing endocarditis that had led to mitral insufficiency. Distinct signs of effusion were not obtainable, and hence it was concluded that the exudate was fibrinous, or if united with serum, the proportion of the latter was not large. The extensive dilatation present was attributed in part to the mitral lesion, and in part to the dilating influence of the pericarditis, whether associated with acute myocarditis or not.

The symptoms in this case were not distinctly those of pressure; respiration was greatly accelerated, to be sure, but there was no cyanosis, no downward displacement of the liver, and no orthopnœa; in short, the symptoms pointed more to disturbance of the nervous system, with consequent rapidity of breathing, than to circulatory embarrassment. The very considerable hepatic engorgement could be very reasonably referred to the free mitral leak and the greatly overstrained right ventricle. This patient ultimately made a good recovery.

On the same day on which I saw the preceding patient, Dr. Josephson asked me to visit a little girl of six, who was also suffering from acute pericarditis. She had passed through an attack of scarlatina in the July preceding, and for the past three or four weeks had been suffering from acute articular rheumatism, which was still present when she came under the doctor's charge eight days before my visit. He had at once recognised an acute inflammatory affection of the heart. Her somewhat fluctuating temperature had averaged about 102° F.

The child's condition when I saw her was as follows: She was sitting in bed, not even venturing to rest against the pillows, breathing 60 or more times to the minute, and during the forenoon of that day her respirations had actually been 90 to the minute. The pulse was very rapid, small, and dicrotic, but perfectly regular. The expression one of patient suffering. Upon removal of the clothing the skin was found hot, dry, and scaly, yet broke out into a perspiration a few minutes afterward upon the child making a little exertion. There was every evidence of capillary and venous congestion. The abdomen was distended and hard, particularly about the waist-line; the thorax was evidently distended to its utmost capacity, the entire front of the chest bulging, and the intercostal spaces more or less smoothed out. The apex-beat was feebly palpable below and a little to the left of the nipple, and there was diffused systolic shock over the body of the organ. Absolute cardiac flatness (Fig. 17) began at the right nipple, passed upward to the first interspace, then downward and outward into the left axillary region, well outside of the visible and palpable apex-beat. Its lower boundary reached at least to the eighth costal cartilage, and the distended sac could be felt in the epigastrium. A rough, blowing systolic murmur was very loud in the mitral area to the left, while the heart-sounds were loud over the base of the organ, the pulmonic second being very banging and slightly split. From the middle of the sternum downward to the ensiform was a grating pericardial friction-sound, which had a simple to-and-fro rhythm not synchronous with either

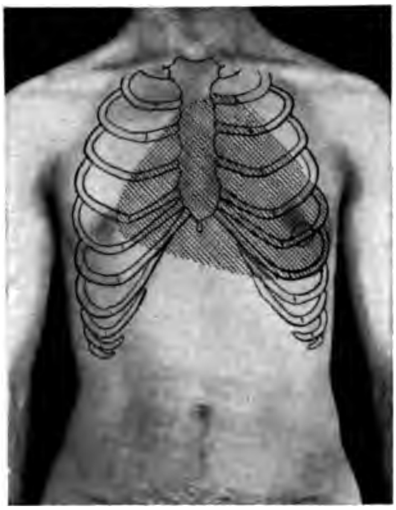


FIG. 17.—ABSOLUTE DULNESS, CASE OF PERICARDITIS WITH EFFUSION.

systole or diastole. At the left base, posteriorly, was a dull patch corresponding with Ewart's dull patch in outline, but the harsh, very hurried breath-sounds were everywhere audible. In front, resonance was impaired beneath both clavicles, and the sense of resistance imparted to the finger upon percussion of the præcordium was remarkably intense.

In this case the diagnosis was also acute pericarditis, but, unlike the foregoing, there was a massive exudate, occasioning very grave pressure-signs. There was also present the same valvular lesion, mitral regurgitation, but there was very strong suspicion of the existence of acute endocarditis, since from the history of scarlet fever in July, with more or less rheumatism subsequently, with no other previous etiological factor, it was not likely that the mitral disease dated back more than six months.

Deglutition gave this little sufferer so much distress that she would hesitate for minutes together before making up her mind to take the proffered medicine or nourishment. In this case the urgency of the symptoms arose from pressure. Dyspnœa was so great that the little thing begged to be allowed to stand up, evidently to relieve the thoracic organs, already much compressed, from still greater pressure by the abdominal viscera forced upward against the diaphragm in the sitting position.

The contrast presented by these two cases was most instructive. In this latter case paracentesis pericardii was advised without delay. It would have been cruel, if not useless, to postpone the operation until trial had been made of cathartics and diuretics. Far better tap first, and administer these afterward.

Suppurative pericarditis generally occasions phenomena of sepsis, but chills, fever, and sweating are sometimes said to be absent (Roberts). When present they are an indication of sepsis, and as such may be a part of the symptomatology of the primary affection as well as of the pericarditis. When the effusion is fœtid, as rarely happens, septic symptoms are most marked, and prostration comes on early and is extreme. In some cases there is nothing in the nature of the symptoms whereby one may determine the purulent character of the exudate.

The symptomatology of *hæmorrhagic* pericarditis depends upon the rapidity with which the effusion takes place, rather than upon its nature. A hæmorrhagic effusion into the pericardial sac during the course of scorbutus, for example, may take place so suddenly that symptoms of pressure and of anæmia rapidly develop. In other cases the effusion is slowly produced, and symptoms manifest themselves gradually or are entirely absent.

In Ebstein's two cases the symptoms were those of pressure, cyanosis, dyspnœa, cough, and pain, but in one the condition was thought to be extreme cardiac dilatation, and its true nature was not recognised until at the autopsy. Although in the second case pericardial effusion was recognised during life, there was nothing in the symptoms to point to the hæmorrhagic character of the exudate.

Course and Termination.—There is no uniformity in the clinical history of pericarditis with effusion. Cases vary widely from each other in the mode of onset, in the course they pursue, and in their mode of termination. An ordinary case occurring during a rheumatic attack may be expected to terminate by absorption in two to four weeks; this happy event may very rarely take place within a few days, the disease having passed through the successive stages of inflammation, effusion, and absorption in less than a week. In other instances the affection manifests a strong tendency to become either subacute or chronic. While in others, again, the disease is characterized by phases of partial absorption and improvement, which are each in turn followed by a recurrence of inflammation and increased effusion (Bauer), until at length the patient is worn out by the persistent and obstinate nature of the disease. These variations depend, no doubt, upon the activity of the etiological agent, and are not at all surprising, for, as every one knows, no disease presents uniformity in its clinical phenomena.

Other conditions besides the activity of the pathogenic agent also determine the course and severity of an acute pericarditis. Its occurrence with or as a sequel to pleurisy or pneumonia is also likely to influence its course and termination, in accordance with the intensity of these latter processes and the degree to which they have undermined the patient's resistance. When pericarditis is the result of chronic nephritis it is very likely to run a slow and latent course.

In children with inflammatory rheumatism the disease is apt to prove persistent, and if it does not destroy life by invading the myocardium, terminates in complete or partial synechia pericardii.

Suppurative pericarditis is a very serious affection, manifesting but little tendency to spontaneous recovery. It is stated, however, that if the pyogenic bacteria be not very virulent, and if life be prolonged for a considerable time beyond the stage of active inflammation, absorption of the more liquid portion of the exudate may take place, the residue becoming cheesy and in time infiltrated with lime-salts. They are eventually transformed into calcareous plates, which may even be so extensive as to inclose the heart in a case of bone-like hardness. I have observed two such cases; in one a calcareous plate the size of a silver dollar was found on the anterior surface of the left ventricle, while in the other, masses of lime completely surrounded the organ. This latter case will be described in the article on Adherent Pericardium. Most cases of purulent pericarditis, unless relieved by surgical interference, pursue a rapid course, and patients succumb more or less speedily to the effects of pyæmia, resulting either from the pericardial or primary affection.

In the form of hæmorrhagic pericarditis, which occurs in the course of scurvy and is observed in the maritime provinces of Russia, the effusion often takes place with great rapidity and destroys life in one or two days. In these cases death seems due in no small measure to the rapidly induced anæmia.

Finally, the course of acute pericarditis with effusion is determined not alone by the intensity of the inflammation, but by the amount of the exudation. If this is sufficient to greatly distend the sac, its absorption is hindered by the tension thus occasioned. In some instances, no doubt, a pericardial exudation is absorbed, and no permanent ill effects remain.

Physical Signs.—*Inspection.*—The degree of information afforded by inspection depends upon the amount of effusion and the conditions residing in the chest-walls. In a child of tender age or a person with a yielding chest-wall a comparatively small pericardial effusion may occasion perceptible prominence of the præcordium, while if the thorax is voluminous, and the costal cartilages have become hard and inelastic from age, it is possible for even an enormously distended sac to produce no visible bulging of the cardiac area. As a rule, however, more or less prominence in this region is observed, while the intercostal spaces look filled out, and the skin covering them appears tense and shiny. The apex-beat is not visible, or but faintly so, and cardiac impulse is feebly diffused or wanting. The apex-beat, moreover, if visible, may be situated lower than normal when the liver is depressed by a massive effusion, and in such a case there may be bulging of the epigastrium.

Cyanosis and distended veins give evidence of circulatory disturbance, while respiratory embarrassment is evinced by hurried breathing and restricted movements of the chest. If copious effusion occasions great pressure, and particularly if this has formed rapidly, the attitude of the patient and the expression of his countenance betray suffering and it may be oppression.

Inspection is an aid to diagnosis, but cannot solely be relied upon, since præcordial bulging in children may be the result of cardiac enlargement without pericarditis.

Palpation.—In great effusion the roughened pericardial surfaces are removed from each other, and hence the hand no longer detects the peculiar fremitus present in the beginning of the process, when the exudate is fibrinous and not serous.

Otherwise palpation serves chiefly to corroborate the result of inspection. The præcordial area may impart a sense of increased resistance from internal pressure, and the normal intercostal depressions are found obliterated. Rarely there is fluctuation. Increased resistance and tension may also be detected in the epigastrium. Older writers were accustomed to attach great importance to an elevation of the apex-beat, which they explained by lifting of the apex of the heart by the effused liquid. Roberts, Ewart, and others regard this as erroneous, believing that what was thought to be the apex-beat is, in fact, the impulse of the body of the heart as it is thrown against the anterior chest-wall by the collection of fluid behind, the apex of the organ being at the same time moved backward and to the left. In cases of extreme effusion the depression of the diaphragm, occasioned by the heavy sac, leads to an actual lowering of the apex-beat (Bauer). The pericardial fremitus present during the inflammatory stage disappears with the occurrence of effusion. In some cases the head of the left clavicle is said by Ewart to be elevated so that the first rib can be felt all the way to the sternum (" first-rib sign ").

The most striking character of the pulse is its want of tension. It is rapid and may be regular or irregular, even intermittent. In some instances *pulsus paradoxus* is present. This is an inversion of what is usually observed during the two acts of inspiration and expiration. Instead of becoming stronger and fuller at the end of inspiration and the beginning of expiration the pulse becomes small and weak, or may even disappear during deep inspiration, becoming again stronger and fuller toward the end of expiration and the beginning of the next ensuing inspiration. *Pulsus paradoxus* is inconstant and is not pathognomonic when present, and possesses therefore only a negative value.

Percussion.—This method of investigation furnishes the only reliable sign of pericardial effusion, since by this means one is often able to determine the presence of so small an amount as 100 cubic centimetres (Bauer), 150 to 200 cubic centimetres (Aparti and Figaroli). That one may understand why so much reliance is to be placed upon percussion, I will consider for a few moments in what way pericardial effusion alters the normal relation of the parts and modifies the area of cardiac dulness.

The pericardium is a closed sac, which is thrown around the heart, being wrapped about the origin of the great vessels above, and attached to the central tendon of the diaphragm below. When fluid is effused into this closed cavity it sinks to the most dependent part, and then creeping upward distends the sac in all directions, pushing aside the anterior borders of the overlapping lungs. The area of absolute cardiac dulness now becomes altered in a striking manner (Fig. 17), and to an extent commensurate with the amount of effusion. Some authorities consider that this alteration of cardiac dulness corresponds in shape with that of the distended sac (Bauer, Sibson), while others attribute it chiefly to the crowding aside of the lung-margins (Duchex, Rotch). My own opinion is that the configuration of this area depends largely upon the anatomical arrangement of the lung-borders overlapping the heart, since when they become retracted by adhesions and in cardiac dilatation, the shape of the resulting dulness is essentially the same, though less extensive, as in pericarditis with effusion. Probably both factors, the shape of the sac and the arrangement of the lung-borders, are responsible for the peculiar outline of the area of dulness observed. This area of absolute dulness or flatness is variously described as triangular, pyramidal, pear-shaped, or pyriform, that of a truncated cone, or " that of a bag of fluid spreading out at the base" (Ewart). Its broad base rests upon the diaphragm, while its rounded apex occupies the upper sternal region. A glance at Fig. 17 shows that the direction of the two side-lines is not the same, being rather more vertical at the right. The right arm of this irregular triangle is shorter and straighter, while the upper or left boundary presents an indentation or concavity soon after leaving the apex, and, sloping gradually downward, joins the base-line at a rounded somewhat obtuse angle.

The shape of this area is by most authors considered very characteristic, although Shattuck is of the opinion that the peculiar feature is not the form, but the fact that the dulness spreads out in all directions. Rosenbach lays stress on the increase or movability of dulness to the right when the patient lies on his right side.

Although Bauer agrees in the statement that the flat area of pericardial effusion may be recognised by its characteristic pear-shaped outline, he nevertheless expresses the opinion that more importance should be attached to the *surrounding zone of relative dulness*, "since, indeed, the absolute cardiac dulness not uncommonly in cases of well-marked effusion shows little or no alteration."

In cases of extreme pericardial effusion the base of this flat area may extend nearly across the anterior surface of the chest, from within, or in some cases even outside the right mamillary line, to a variable distance outside the left mamillary or to the left anterior axillary line. In consequence of the depression of the left lobe of the liver caused by the weight of the distended sac, the inferior margin of this area may reach as low as the sixth, or in extreme cases even the seventh left intercostal space, while its broad conical apex may extend as high as the level of the second costal cartilage or the first interspace.

When this flat area has attained such proportions it is usually not difficult to determine the nature of the case. Yet, since a greatly dilated heart may also crowd aside the lungs and occasion a similar extension of the area of cardiac dulness, error can only be avoided by attention to the following points: (1) When the pericardial sac is distended by fluid its left latero-inferior boundary extends beyond the situation of the apex-beat, and hence this latter, as determined by auscultation, is found situated within and above the extreme left lower angle of cardiac dulness. In dilatation of the heart, on the other hand, it is the organ itself which determines the dulness, and therefore the apex-beat corresponds with the outer and lower limit of cardiac dulness. Leube lays great stress on this point in the differential diagnosis of these two conditions. (2) In cases of pericardial effusion the line of demarcation between the area of flatness and surrounding pulmonary resonance is very abrupt, while in cardiac dilatation the transition from flatness to resonance is much less pronounced. Bauer and Sansom both attach great importance to the abruptness of this transition in cases of pericardial effusion. At the same time one should not forget the fact that occasionally the distended sac may be overlapped by the lungs, and therefore absolute dulness may shade off through a surrounding zone of comparative dulness into full pulmonary resonance.

When pericardial effusion takes place the first change noticeable upon percussion is the development of a small triangular area of dulness in the fifth right intercartilaginous space, or, as the Germans term it, in the *cardio-hepatic angle* (Fig. 18). This sign, first described by Rotch, and sometimes called *Rotch's sign*, is due to the fact that when effusion collects it first distends the sac at its *lower right corner*, occupying the space between the curved inferior margin of the heart and the upper line of hepatic dulness immediately to the right of the lower end of the sternum. Ewart and Ebstein have also directed attention to the occurrence of this small triangular dull patch.

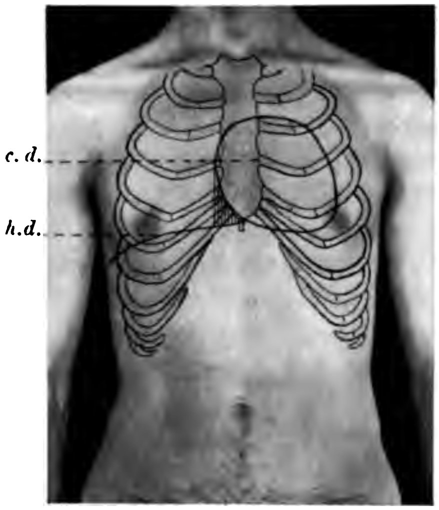


Fig. 18.—Rotch's Sign of Beginning Pericardial Effusion.

Dulness in shaded area or cardio-hepatic angle: *c. d.*, cardiac dulness; *h. d.*, hepatic dulness.

Normally, the outer boundary of cardiac dulness over the right auricle presents a curved line, passing from the level of the third costal cartilage downward and outward, and, after crossing the fourth cartilage, passes inward as well as downward to join the inferior margin of the right ventricle. (See Fig. 2.) In the formative stage of pericardial effusion, on the contrary, the right border of heart-dulness is no longer curved, but passes directly downward, joining liver-dulness at an abrupt angle. Rotch's sign is therefore of great value in determining the beginning of pericardial effusion.

One occasionally sees statements to the effect that an alteration in the extent of cardiac dulness depending upon the patient's position makes strongly for pericarditis with effusion. This is nevertheless a very untrustworthy sign, since, if the heart is greatly enlarged, it may fall away from the anterior chest-wall in the dorsal decubitus with consequent diminution of cardiac dulness, and in the erect posture again approach the anterior parietes and occasion a corresponding increase in the heart's area. No inconsiderable danger of fatal syncope sometimes attaches to the patient's change of position from the recumbent to the erect, and since the sign just alluded to is of but slight value, one is hardly justified in thus subjecting a patient with pericarditis to the risk of sudden death.

Auscultation.—As a rule, the pericardial friction-sound of the first stage disappears with the occurrence of effusion, to reappear after absorption has again allowed the roughened pericardial surfaces to come in contact. Nevertheless all writers agree in stating that the persistence of the friction-sound is not incompatible with a considerable amount of fluid, even as much as a quart (Cejka), in the pericardial sac. The explanation of the non-disappearance of the friction-sound in such cases is found in the presence of adhesions over the body of the heart, which prevent the separation of the epicardium from the pericardium, and force the fluid to the dependent parts surrounding, or in failure of the sac to be completely filled. In other instances the friction-sound becomes faint when not wholly inaudible.

Any alteration that takes place in the heart-sounds affects their intensity rather than their quality, since they have to be transmitted to the ear from a distance proportionate to the recession of the heart from the chest-wall and through a layer of fluid. If the effusion is massive and fills the sac to its utmost capacity the cardiac tones may be inaudible, but this is so rare that I have never observed a case in which they were wholly absent. In most cases the sounds at the apex are feeble, but not wanting, while at the base they are heard more clearly. The pulmonic second sound is accentuated and the aortic sound is diminished.

In a recently observed case of extensive pericardial effusion in a child the cardiac impulse, the sounds, and a previously existing endocardial murmur all remained distinct over the body of the organ, and were attributed to the presence of adhesions that had forced the fluid to the side of the sac.

Secondary Physical Signs Referable to the Lungs.—Valuable information of the existence of pericardial effusion may also be obtained by examination of the lungs. The retraction and compression of pulmonary tissue occasions a loss of normal pulmonary resonance in the neighbourhood of the sac. In the left infraclavicular region percussion elicits Skodaic resonance, or if the compression be very great, impairment of the note. Both Pins and Ewart have called attention to certain changes discoverable by percussion and auscultation at the posterior base of the left lung. The "Pins' sign" is dulness and bronchial breathing in the left infrascapular region, which, upon the patient leaning forward, give way to tympanitic resonance, crepitant râles, and finally vesicular breathing. This dulness and bronchial breathing can hardly be attributed to pleuritic effusion, if one bears in mind the curved upper line of the dulness in this latter affection.

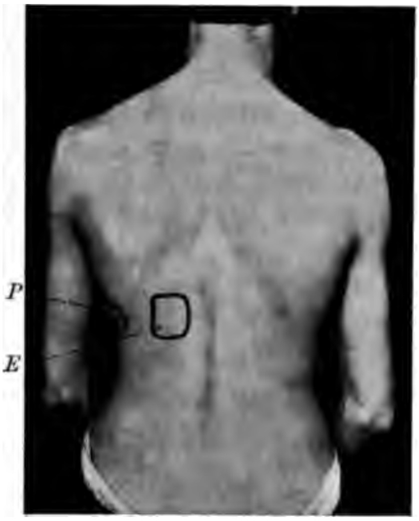


FIG. 19.—LOCATION OF PULMONARY CHANGES IN PINS' (*P*) AND EWART'S (*E*) SIGNS OF PERICARDIAL EFFUSION.

Ewart has described a posterior patch of dulness in cases of extensive pericardial effusion situated at the base, and which, extending from the spinal column outward nearly or quite to a line corresponding with the internal border of the scapula, turns abruptly upward at a right angle, and, after reaching the level of the ninth or tenth dorsal spine, again turns sharply inward to reach the side of the vertebral column (Fig. 19). Over this patch breath-sounds are wanting and voice-sounds are feeble. Occasionally a similar dull area may be found to the right of the spine. Ewart attributes this sign to " altered dorsal relation of the liver." Immediately below and to the left of the tip of the left scapula, especially in children (Sansom), is a dull patch of about 2 inches diameter, in which are bronchial breathing and bronchophony or ægophony. Ewart has also directed attention to a somewhat inconstant sign consisting of a small area of bronchial breathing in the right mamillary line, between the right nipple and the upper surface of hepatic dulness.

Diagnosis.—As a rule it is not difficult to determine the existence of pericardial exudation when this is abundant, but it is not easy nor always possible to determine its nature. If the pericarditis arises in the course of articular rheumatism, and signs of distention of the sac present themselves, the exudation is probably sero-fibrinous. Of 324 cases of fatal pericarditis occurring at the Berlin Charité between 1866 and 1876, and which were analyzed by Breitung (Eichhorst), sero-fibrinous was found 108 times, as against 24 of purulent and 30 of hæmorrhagic exudation. Furthermore, the nature of the primary affection to which the pericarditis is attributable is of diagnostic significance, since it is likely to determine the nature of the exudate. If the inflammation occurs in the course of septicæmia, or results from extension of empyema, or from the perforation of a gastric ulcer, from a perforating wound, etc., it is likely to be purulent. The appearance of distinct septic phenomena, rigors, and an irregular intermittent fever, profuse perspirations, great and rapidly increasing prostration, a dry, coated tongue, diarrhœa, etc., warrants the diagnosis of pyopericardium.

If signs of fluid distention of the sac develop rapidly in the course of scorbutus or purpura hæmorrhagica or some other dyscrasia, as cancer, and particularly if accompanied by pronounced and rapidly increasing anæmia, the probability is in favour of a hæmorrhagic pericarditis. Inasmuch, however, as such theoretical distinctions in the symptomatology of the three varieties are not always clearly defined, a differential diagnosis is often only possible by means of exploratory puncture.

Differential Diagnosis.—Before considering the differentiation of fluid-collection within the pericardium from certain other conditions with which it may be confounded, it is well to speak of the difficulty of diagnosis arising from pulmonary emphysema and old adhesions that date from a previous attack of pericarditis. Emphysema may prevent the lung-margins from being crowded aside, and hence the characteristic area of cardiac flatness may not exist. In such an event reliance must be placed on the outline of deep-seated dulness, and this failing, absolute diagnosis is hardly possible. When old adhesions exist over the front of the heart they force the exudate to accumulate at the sides and bottom of the sac or to be pent up posteriorly. Accumulation of fluid along the lateral and inferior margins causes increase of dulness in these situations, its triangular outline being fairly well maintained. In addition, the cardiac impulse and tones, together with the friction-rub, remain both palpable and audible over the body of the organ.

When effusion is confined to the posterior aspect of the sac its recognition is most difficult, if not impossible. In such a case one must rely mainly on the symptoms of (1) inflammation, as pain and fever, and (2) of a deeply situated collection of fluid and pressure on the œsophagus and bronchi, dysphagia, and dyspnœa. The last two symptoms are very suggestive of exudation into the posterior portion of the sac. Massip calls this an Encysted Pericarditis with retrocardiac effusion, and says its symptoms are so obscure that one can do no more than diagnose the pericarditis without being able to decide whether effusion is present or not. He thinks that dulness at the left posterior base with muffling or absence of heart-tones in this region, together with signs of pressure on the œsophagus and of active pericardial inflammation, are strongly suggestive of a posterior effusion.

Dilatation of the heart is the condition which most often has to be differentiated from pericardial effusion. The enlargement of the organ causes retraction of the lung-borders and an area of dulness very like that of effusion, and if the heart-tones and impulse are very feeble, from fatty degeneration, a precise diagnosis may under certain circumstances be extremely difficult. The main point on which reliance is placed is not, as sometimes stated, greater distinctness of heart-shock and sounds in dilatation as compared with effusion, but is the position of the apex-beat with relation to the outer and inferior margin of dulness (see page 77). Theoretically this is very fine, but every experienced clinician knows that in many cases it is impossible to say definitely whether they correspond or not on account of the indistinctness and diffuseness of the cardiac impulse. I once saw the late distinguished von Ziemssen, one of Germany's most skilful clinical teachers, make a mistake in just such a case. The patient, who was *in extremis*, was presented to the class as an instance of massive pericardial exudation. There was a large triangular area of absolute cardiac dulness over which impulse was wanting, and heart-sounds were scarcely audible. Moreover, râles of acute pulmonary œdema rendered auscultation highly unsatisfactory. The autopsy, next day, disclosed a fatty and extremely dilated heart, but no effusion, and von Ziemssen took the occasion to teach a most instructive lesson on the difficulties of differential diagnosis.

Should it prove impossible to locate the apex-impulse in the recumbent posture, the patient may be slowly and cautiously lifted into the erect position in the hope of the heart gravitating forward, and thus declaring the situation of its apex. If this not entirely safe procedure fails, then aid may sometimes be derived from careful study of the pulse, which in pericardial effusion is said to occasionally be relatively stronger than the feebleness of the heart-sounds would lead one to expect.

This is also a theoretical point which I have never found to stand the test of practical experience. When a pericardial exudation is massive, as well as when extreme dilatation simulates effusion, the equilibrium of circulation is lost, the veins are overfilled, and the arterial system is empty, so that, as a matter of fact, the pulse of extensive effusion is small, weak, and rapid. Should all attempts to arrive at a differential diagnosis fail, then, as suggested by Leube, we may have recourse to digitalis and other therapeutic measures in the hope of clearing up the condition. In dilatation proper treatment may revive the flagging heart and restore the apex-beat and heart-sounds, while in pericarditis it may cause absorption of fluid and a reappearance of pericardial friction.

Pleurisy with effusion may be mistaken for pericarditis, or rather a pericardial may be considered a pleuritic effusion. There is much similarity in the pain, fever, and dyspnœa, as well as certain pressure-effects, but error is avoidable by attention to the following points: (1) In pleuritic effusion there is a curved line of flatness which extends from the back around the side to the front, which line usually shifts with change in the patient's position. (2) A left-side effusion (the only one likely to lead to error) pushes the heart over to the right of the median line. (3) Breath-sounds at the left base are diminished or absent, instead of exaggerated or bronchial, as in pericardial effusion. (4) Dysphagia is very rarely if ever present in pleurisy. (5) In pericarditis there is usually a history of rheumatism or other acute infectious process to lead to cardiac involvement. In an adult a differential diagnosis between these two affections is seldom difficult, but I have seen young children in whom it was at first not at all easy to say which was the process, owing to the great compression of the left lung and consequent extent of dulness laterally and behind.

The foregoing are the only two affections likely to mislead; yet the careless, and still more the inexperienced, may mistake for pericarditis a number of conditions that occasion increase in cardiac dulness upward and to the left. These are mediastinal tumours, which crowd the lung-margins aside and displace the heart, and pulmonary tuberculosis or old pleuritic adhesions, which cause permanent retraction and fixation of the anterior border of the left lung. Error ought to be avoided, however, by due attention to history, symptoms, and physical findings. The history is that of insidious commencement and slow course—symptoms are those of a chronic process without fever, except, of course, in the case of pulmonary tuberculosis, when there is characteristic sputum to act as a guide—and as regards clinical findings, the dulness lacks the distinctively triangular shape of pericarditis with effusion. Time settles the diagnosis beyond question.

Prognosis.—This depends upon the nature of the exudate, the rapidity of its formation, its amount, the effect, both of inflammation and resulting effusion upon the myocardium, the existence of complications, as acute or chronic endocarditis, Bright's disease, tuberculosis, etc., and finally, the age and vitality of the patient.

Suppurative pericarditis, unless recognised and treated surgically, is very likely to prove fatal, and yet, as previously stated, if of rather a benign type the fluid portion of the exudate may ultimately become absorbed, leaving a cheesy, and at times a calcareous, mass behind.

Pericarditis hæmorrhagica acuta may destroy life within a few days. When the malady is chronic, its prognosis is essentially that of the scorbutus or other primary affection.

An effusion of whatever nature that forms rapidly and to a large amount is always serious, because time is not allowed for the system to adjust itself to the altered conditions. Such a case may speedily prove fatal. If the inflammation extends to the myocardium, or if this latter has undergone previous degeneration, the heart-muscle will be ill prepared to sustain the pressure exerted by the fluid confined within the tense and resisting sac. Serious circulatory and respiratory embarrassment, or the possibility of fatal syncope, renders the immediate prognosis most grave.

Acute endocarditis is a serious complication, and the existence of a chronic valvular lesion occasions a degree of gravity which might not be the case if the pericarditis existed alone. If the disease occurs in a person with acute or chronic nephritis, or if pulmonary tuberculosis coexists, the patient is hardly in condition to successfully cope with the occurrence and long duration of an exudative pericarditis. Moreover, coming on as it does toward the end of chronic nephritis, the pericarditis contributes largely to the fatal result.

Finally, emphasis has been repeatedly laid on the serious nature of pericarditis in children, owing to the frequency with which it implicates the heart-muscle and the strong likelihood of its leading to cardiac dilatation. In children, therefore, the immediate, as well as the ultimate prognosis, is serious. In old people and those enfeebled by some chronic malady that has brought them to a state of cachexia there is small likelihood of the patient surviving until time can bring about absorption of the effusion.

In all cases, even when death does not result directly from the acute inflammatory or exudative process, there is a possibility of the heart being crippled by inflammatory damage to the myocardium, or by partial or total obliteration of the sac. Of the 324 cases analyzed by Breitung, there were circumscribed adhesions in 111, and complete adhesion of the pericardium in 23. The remote prognosis depends upon the richness of the exudate in fibrin; in such the likelihood of adhesions is the greater. Pericardial inflammation, therefore, of whatever nature, should never be looked upon as a trivial complaint.

Treatment.—We possess no means of arresting an attack of pericarditis, and therefore we must content ourselves with endeavouring to combat the rheumatism or other affection in the course of which pericardial inflammation occurs, in the hope of preventing the latter. If, nevertheless, the sac becomes involved, we must strive to lessen the severity of the process, and this failing, to relieve symptoms and sustain the powers of life until the disease comes to a natural termination.

Inasmuch as we are powerless to abruptly terminate pericarditis, and it occurs most often in the course of articular rheumatism, every possible effort should be made to cut short or mitigate the intensity of the rheumatic attack. This is no place to discuss the treatment of the latter affection, yet I wish to record my confidence in the salicylic-acid treatment, especially in methylsalicylate, both locally and internally. If this is not a specific, we at all events possess nothing better, and must await the time when definite knowledge of its nature may supply us with an efficient weapon against rheumatism.

The importance of *physical rest* cannot be too strongly insisted upon whenever fever or other rheumatic symptoms make their appearance. This is particularly wise in the case of children who have an old-standing valvular lesion. Such children ought to be seen by the family doctor whenever sore throat or other suspicious symptoms arise. Physical exertion by increasing the force and frequency of cardiac contractions, tends not only to intensify pericarditis when it is already present, but may even determine its development in the same way that use of a rheumatic joint may aggravate the arthritis. If an adult is unwilling to submit to rest, he should be informed of the possibility of endocardial or pericardial inflammation, and thus perhaps be induced to take proper care of himself.

When pericarditis once sets in, measures are indicated to lessen its intensity. Vesication of the præcordia was once extensively used for this purpose, and there are still many physicians who believe in the efficacy of this treatment in the initial stage. Personally, I am sceptical of the influence of blisters in this regard, and believe that milder measures will do just as much good, while at the same time saving the patient from the pain and discomfort of a large blister. The real benefit of vesication, in my opinion, is found in the relief of pain, and therefore I think it is preferable to adopt small blisters, a fresh one being applied so soon as the previous one has filled. This is the plan advocated by Caton, because of the ease to pain thus afforded. I have had no great experience with this mode of treatment, because I prefer the application of cold to the cardiac area. I know that counter-irritation often eases the suffering occasioned by inflammation of serous membranes, and therefore advise that if cold is not well borne and the intensity of the initial pain seems to call for some measure of the kind, that this be a sinapism made of English mustard by the nurse, and that it be left on until the skin becomes thoroughly reddened. When the mustard-draught is removed, its place may be supplied by a poultice or by hot fomentations. Moist heat to the præcordium gives great relief in some cases, and is much extolled. Roberts applies 2 or 3 leeches over the heart in suitable cases, but as a rule finds poulticing in the early stage gives positive relief to pain.

Lees is a strong advocate of the continuous use of the *ice-bag*, and asserts that it not only gives comfort by alleviating pain and palpitation, but tends to mitigate the severity of the inflammatory process. This mode of treatment has always appealed to me as rational, and in all cases in which I have seen its use faithfully tried it has appeared to be very comforting and agreeable. The ice-bag must be light, so as not to oppress the patient by its weight, and should be held in place by a cord passing around the neck. As the sufferer is usually in a semi-recumbent posture, the bag, thus suspended, rests lightly on the præcordia without danger of slipping off. Furthermore, the ice-bag must not be allowed to rest against the bare skin, as it is apt to occasion irritation, but a small piece of dry, thin cloth is to be interposed between the surface of the chest and the bag. In this manner the bag is generally well borne, when before it could not be endured. Children are sometimes uneasy at first, yet if the application of cold is firmly insisted upon, they not only learn to tolerate it, but actually find it soothing. Should idiosyncrasy render an individual absolutely intolerant of cold, then it may be replaced by poultices. Hot fomentations are objectionable on account of the liability of their wetting the clothing, and of a chill when the cloths are changed. In the employment of the poultice due attention should be paid to the principle that to be efficacious it must be hot, not merely warm, and must be replaced by a fresh one so soon as it grows cool. When at length poultices are discontinued the surface of the chest must be covered by a layer of cotton or flannel.

Treatment in the inflammatory stage is largely symptomatic, and in most cases something more than either heat or cold is required to allay pain and restlessness. In mild cases an anodyne liniment, as belladonna, chloroform, or one containing morphine, may suffice, and should be tried before recourse is had to internal medication.

When pain and restlessness are severe nothing is so serviceable as opium in some form. In the case of adults a hypodermic of morphine is the best; to children it is far better to give the remedy by the mouth. Their well-known susceptibility to the drug makes it advisable to try the effect of codeine before resorting to opium or morphine. In some cases it will be found that a combination of codeine and sodium bromide will act efficiently and render more powerful remedies unnecessary—a consideration to be always borne in mind with children.

Besides allaying pain and quieting the little sufferers, these agents tend to lessen the violence of heart-action—a desideratum of importance—and to promote sleep. *Insomnia* is often very troublesome, and when not overcome it contributes greatly to the patient's nervousness and inability to bear well the strain of a protracted illness, which, like pericarditis, makes great demands on the patient's powers of endurance. An opiate steadies the nerves, and if it does not too greatly disturb digestion and secretion I believe it cruel to withhold it in cases characterized by the foregoing symptoms.

Cough is an annoying feature in some cases, and when this is so it affords an additional reason for the administration of codeine or morphine. A preferable remedy, however, is heroin, which to an adult may be given in the dose of $\frac{1}{12}$ grain, and to children in proportionately smaller amounts. It not only allays cough efficiently, but is devoid of the unpleasant after-effects of morphine.

Nausea and *vomiting* may in some cases tax medical skill to the utmost, and, as in a recently observed instance, defy all attempts to allay them. In such an event it becomes necessary to stop oral administration of food and medicines, and to rely on enemata in the hope of the stomach becoming quiet.

Fever does not always require antipyretic treatment, but should it persist at 102° F. or higher it may be reduced by sponging.

In this early stage *rapid, violent action of the heart* is often present, and seems to call for quieting measures. Digitalis does not appear to me to be indicated, for tachycardia is now not a manifestation of weakness, but of irritation, and in my experience the heart does not bear kindly attempts to slow it by digitalis. Neither should aconite or veratrum be prescribed for this purpose, since they are too depressing, and the heart is likely to need all its reserve force before the struggle is over. I believe no therapeutic measure is more efficient in quieting the organ than an ice-bag worn continuously.

The *routine administration of digitalis* is objectionable in any form of cardiac disease, and in pericarditis is especially so. The real indication for its use in this affection is not merely rapidity of the pulse, but feebleness, together with rapidity. Therefore, should the unchecked tachycardia begin at length to tell on the heart, or should the organ furnish signs of dangerous dilatation with scantiness of urine and other evidences of visceral congestion, then it is well to prescribe digitalis. The hypodermic administration of digitalin does not seem to me so reliable or effective as the internal use of a fat-free tincture, or of the infusion in moderate doses, 10 drops of the former and a tablespoonful of the latter to an adult, and to a child a proportionately smaller amount every four to six hours.

Strychnine is a heart-tonic which cannot be dispensed with in this stage. Its dose does not need to be large at first, perhaps $\frac{1}{60}$ if the patient is grown, 3 or 4 times a day. It may be given by the mouth, but under the skin is preferable, since its action is more direct and powerful.

There is always a more or less pronounced tendency to congestion in pericarditis, the liver feeling the brunt of the attack, and hence being usually palpable. Consequently it is well to relieve visceral and portal congestion by a mild daily laxative, calomel or a saline aperient water. Vigorous depleting measures in the inflammatory stage are harmful, however, rather than beneficial, and should be reserved against the time when fluid accumulation occasions distress.

Food must be light and nutritious, consisting largely of milk and nourishing soups and broths, into which a raw egg has been dropped. If fever is not high, and the patient's condition demands heartier food, this may be given in the form of chicken, raw oysters, a bird, or a small piece of carefully broiled beefsteak, with toast or light biscuit. An occasional eggnog is also excellent. It is better to feed these patients often and in small amounts than to supply them with a hearty meal only 3 times a day. Pure or slightly acidulated water should be given freely so long as fever and thirst are present. It also promotes excretion and protects the kidneys from the injurious effects of toxins. In most cases skilful nursing is far more necessary and beneficial than medication.

Treatment in the Stage of Effusion.—With the appearance of exudation and abatement of active inflammation, symptoms of pressure supervene and demand attention. The object of management is now threefold: (1) to restrict the rapidity and amount of effusion, (2) to aid the heart in its attempt to maintain circulation, (3) to promote removal of the exudate by absorption or otherwise.

In my opinion, it is very doubtful if we possess any means of limiting the amount of effusion, since we have no criterion by which to estimate the efficiency of measures employed in any given case. A single large blister or a succession of smaller ones over the præcordia is recommended by some authors; but what assurance have we that the withdrawal of serum in anywise diminishes the amount poured out in the sac? As a matter of fact, the quantity of the exudate, and the rapidity of its formation, are determined by the intensity of the inflammation. If, therefore, we are to lessen the amount of effusion, we must restrain the activity of the inflammatory process. Measures to this end have already been discussed, and although undoubtedly they should be employed, their utility is open to doubt. I pass, therefore, to the consideration of the second object of treatment.

In some cases effusion takes place with such rapidity and induces such urgent pressure-symptoms that surgical interference has to be resorted to without delay. More often, however, indications of pressure appear slowly, and time is afforded for a trial of medicinal treatment.

Absolute rest is now imperative, and the patient, if old enough, must be advised of the necessity of refraining from any sudden movement, lest it occasion fatal syncope. He should be supported in a semi-recumbent posture by a bed-rest or pillows, and he must not be allowed to sit up to take nourishment or medicine. He should be disturbed as little as possible for the purpose of examining the heart.

The state of the circulation, as shown by pulse and venous engorgement, is to be carefully watched, and so long as the quality and rate of the pulse remain good, strychnine may be the only heart-tonic required. So soon, however, as the pulse shows dicrotism or irregularity in force, size, and frequency, tincture of fat-free digitalis must be ordered in doses suitable to the age of the patient—to an adult 10 drops every four to six hours.

The daily quantity of urine should be accurately noted, and in these cases a pronounced falling off of its amount is an indication for digitalis, even though the pulse remains fairly good.

Careful examination of the liver will always detect more or less engorgement of this organ. Palpation of the liver is often painful or unsatisfactory by reason of abdominal distention, but we know by experience and deduction that stasis within the portal system is taking place, and hence hydragogue cathartics form an important, nay an indispensable, part of our therapeutic measures in this stage. By depleting the portal system catharsis aids in maintaining the venous circulation. Instead of weakening the patient, it adds materially to his comfort by lessening epigastric pain and relieving abdominal pressure.

Sleep should be induced by a hypnotic, for nothing will more surely tend to exhaust the nervous system than insomnia. I have sometimes found that the addition of a ¼ or ½ grain of codeine to a sulphonal powder insures the action of the latter. At this stage morphine or a preparation of opium is to be given with very great caution. It may be indicated by dyspnœa or restlessness, but actual danger attends the administration of the drug, through its depression of the respiratory centres. If it be given to allay the dyspnœa, atropia should always be added to the morphine, because of its well-known effect in deepening respiration.

The passive congestion within the abdominal cavity impairs digestion and lessens absorption, yet nourishment is imperatively demanded, both by the nerve-centres and heart-muscle. Only such food should be given as can be easily assimilated, and as but a small amount should be taken at a time, it should be concentrated and highly nutritious. Some of the prepared foods will now be found to be of great service.

When by the subsidence of the pyrexia, if that has existed, it is judged that the active inflammatory process has ceased, or when repeated examinations of the heart indicate that the amount of effusion is stationary, the query naturally arises, What means are to be employed for its removal? Shall an attempt be made to promote this by absorption, or shall paracentesis pericardii be performed? This brings up the question, is absorption of the effusion possible? Clinical experience certainly gives an affirmative answer. Theoretically, absorption of the exudation may be hindered or prevented by an abundant coating of fibrin over the surface of the pericardium whereby the fluid cannot reach the lymphatic spaces, or in consequence of great venous stasis the lymphatic vessels may be surcharged, and the flow in them be too sluggish to promote active absorption. Nevertheless, particularly in rheumatic cases, experience affords abundant proof of the frequent, even rapid, absorption of extensive pericardial effusion.

Therefore, I believe the physician is culpable who refuses to make the attempt at least to carry off the fluid by a resort to diuretic and cathartic remedies. A great degree of venous stasis often neutralizes the effect of diuretics until congestion within the renal veins has been relieved by resort to hydragogue cathartics. The two classes of remedies should be conjoined therefore. In my opinion, the *infusion of digitalis* affords the best means of establishing free diuresis. A tablespoonful to begin with may be administered to an adult every four hours, while the patient also receives daily some unirritating cathartic, capable of inducing a number of copious watery stools. I have seen truly astonishing results follow such a plan of treatment. This is well illustrated by the case of a boy of seven years with mitral regurgitation of rheumatic origin, in a state of perfect compensation, who had been under occasional observation for a year.

On May 9, 1899, he developed what appeared to be a mild case of follicular tonsillitis, for which he received appropriate treatment. Three days later he was reported not so well, and at my visit that same day he was found to have a temperature of 101° F. He complained of vague pains in the knees, which were not reddened or swollen, but showed an erythema. As the attack was undoubtedly rheumatic, salicylate of soda was ordered, and as the boy lived in a suburb he was put in charge of a local physician. May 28th I was asked to see him again, when a præcordial fremitus, which disappeared upon pressure, and a soft to-and-fro murmur, quite different from his endocardial one, with which I was so familiar, left no doubt of the existence of an acute pericarditis. The attending physician stated that these friction-murmurs had developed a few days previously. By June 5th the actual increase in the area of cardiac dulness gave evidence of the occurrence of effusion, though the cardiac impulse and friction-sounds still persisted over the body of the organ. Pyrexia was moderate, pulse 120, of good quality, and respirations were accelerated. An ice-bag had been worn most of the time since May 28th, although pain had at no time been a very marked feature. From now on the exudation increased steadily in amount until, by June 28th, the extreme limits of cardiac dulness reached from just within the right nipple, quite to the left axillary line, far outside the easily located apex-beat (see Fig. 20).

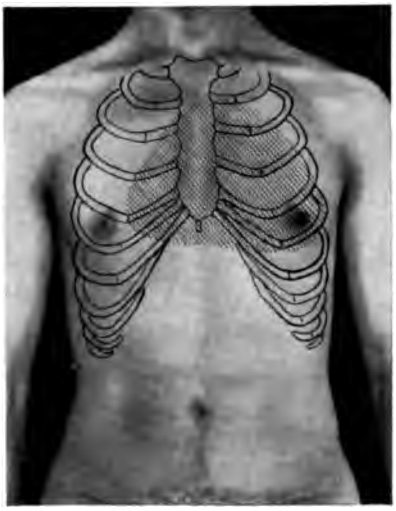


FIG. 20.—APEX-BEAT AND AREA OF CARDIAC DULNESS IN CASE OF PERICARDITIS WITH EFFUSION.

The persistence of the pericardial rub, somewhat less intense, to be sure, together with palpable cardiac impulse over the body of the heart, and the distinctness of the heart-sounds and of the mitral regurgitant murmur, were thought to indicate the existence of adhesions on the anterior surface of the organ and to have prevented the effused fluid from covering over the heart. That the effusion was considerable was evinced by the great distention of the sac laterally and downward, by great pressure upon the lungs, and pronounced circulatory embarrassment. There were marked cyanosis, distention of the superficial veins, great enlargement and tenderness of the liver, and slight ankle œdema. Respirations were 48, pulse 146, small, and dicrotic, but still regular. Salicylate of soda had been discontinued upon appearance of the effusion, lest it might too greatly depress the heart, and as the scanty urine was highly acid, bicarbonate of soda and citrate of potash had been substituted. As digitalis and mild cathartics had failed to appreciably diminish the amount of effusion, surgical interference was decided upon.

The selection of the site of puncture was left to me, and, believing that in consequence of adhesions over the front of the heart the exudation could be most surely reached at some lateral point, I selected the fifth left interspace, between the apex-impulse and the outer margin of flatness (Fig. 21). Accordingly the surgeon introduced his trocar in that situation and obtained fluid. This was distinctly bloody in appearance and so surprised the operator that after permitting an ounce or two to flow he withdrew his cannula for the purpose of discussing the significance of the blood. We concluded it was a hæmorrhagic effusion, and advised a fresh tapping. This was now objected to by the parents on the ground that once at a time was enough, and a repetition of the procedure was deferred. It was never repeated, however, and because of the following considerations:

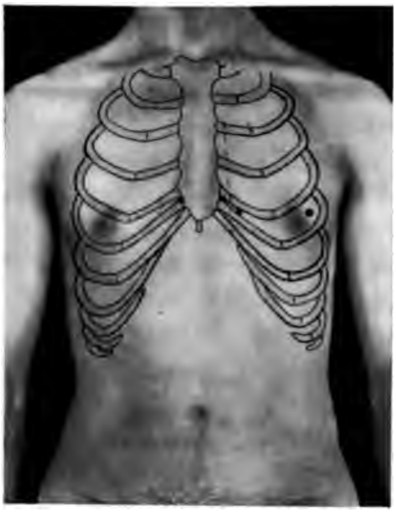


FIG. 21.—THE VARIOUS SITES FOR PUNCTURE IN PARACENTESIS PERICARDII.
Dotted line indicates course of internal mammary artery.

The great obstacle to absorption was believed to lie in the enormous congestion within the portal system, and the boy's distress was due not so much to pressure from the effusion as to the extreme stasis within the abdomen. Tapping the pericardium might relieve the heart, but with all the conditions present in the mitral regurgitation for the maintenance of hepatic engorgement it could not materially improve the state of things in the portal system. Consequently, with the heart-muscle showing no very threatening signs of failure, it was thought best to make one last vigorous onslaught on the stasis within the hepatic vessels. Accordingly a drachm of the saturated solution of Epsom salts was ordered hourly until it began to exert effect. At the same time a drachm of the fresh infusions of digitalis made from English leaves was prescribed every four hours. The results were astonishing. Several doses of the magnesia sulphate were taken next morning without any very marked effects upon the bowels, but instead the kidneys began to act, and in the next twenty-four hours diuresis amounted to something like 8 quarts. Not only did the patient's dyspnœa lessen, but the area of cardiac dulness began promptly to diminish in size, the liver became softer and less tender, and the patient's improvement was clearly noticeable even to his parents. The salts were continued daily, though with gradually diminishing amounts. After two or three days sirup of squills was added to the infusion of digitalis, and the progress towards recovery continued without interruption. This little patient lived nearly three years, and then died from a fresh pericarditis, with signs of dilatation, but not effusion.

Some are sceptical concerning the efficacy of medicinal treatment in promoting absorption, basing their objections on the fact that absorption sometimes sets in spontaneously, even after the pericardial effusion has remained stationary for some time. Nevertheless, in this case, the change for the better began so soon after the administration of the magnesium-salt that I believe it can be justly regarded as an instance of *propter hoc*, not *post hoc*. Unfortunately, the result of therapeutic measures is not always so brilliant as was this. The effusion persists in spite of hydragogue cathartics and diuretics, or the exudation takes place so rapidly and copiously that it threatens to overpower the heart before time is allowed for remedies to exert their effect. In either event the pericardium ought to be tapped, and it is best not to delay. It seems to me there can be no question concerning the indication for paracentesis in such cases, but early in the disease I see no call for surgical interference so long as alarming pressure-symptoms do not supervene.

Sites for Puncture.—Properly performed, this operation is safe, and as it is likely to afford prompt relief, one should always stand ready to tap whenever such intervention is indicated. The indications are not always found in dyspnœa, cyanosis, and rapidity of the pulse; these symptoms are present in all cases of pericardial effusion of considerable amount, but indications are present whenever the heart shows evidence of dangerous weakness by syncopal attacks and intermittence, or when sufficient time having been given for spontaneous recovery, or for absorption through medicinal treatment, these do not take place. Paracentesis being decided on, it only remains to select a suitable point for puncture. Various sites (see Fig. 21) are recommended, and all aim at reaching the fluid most readily without fear of wounding the heart, internal mammary artery, or other structures.

The point most usually recommended is in the fifth left intercostal space at a safe distance from the internal mammary artery. As this passes downward from ¼ to ½ inch from the edge of the sternum, the needle may be introduced either very close to the sternal border, so as to be between it and the vessel, or at the outer side of the artery 1 inch or more from the bone. Rotch prefers the fifth right interspace close to the sternum, since at this point the sac is sure to be distended, even should the amount of fluid prove smaller than is anticipated. Shattuck has punctured in the fifth left space, 1 to 2 inches outside the nipple-line, just within the left lateral border of cardiac dulness, where, as a matter of fact, I advised tapping in the case narrated, although at the time I was not aware of Shattuck's recommendation. The objection urged against this site is the possibility of wounding the pleura at this point. This is, of course, a cogent reason, yet if the sac is greatly distended it will have pushed the border of the left lung well aside, and the pericardium will occupy the region normally filled by the lung. In my case fluid was readily reached, and the surgeon was confident he did not touch the pleura. Another point at which the sac can often be safely pierced in cases of extensive effusion is in the angle between the left margin of the xiphoid cartilage and the adjacent costal cartilages. The fluid tends to gravitate to the bottom of the sac, and consequently weighs down the diaphragm and left lobe of the liver, so that if the needle is thrust upward and backward there is very little danger of wounding the diaphragm. For additional particulars concerning paracentesis, as well as incision of the pericardium, the reader is referred to works on surgery.

Whether all the effusion possible is to be withdrawn, or whether only a portion, sufficient to lessen the pressure and favour subsequent absorption of the remainder, is a matter that must be left to the judgment of the operator. Personally, I advocate the removal of the whole or of as much as can be taken without danger of the heart coming in contact with the point of the needle.

Theoretical considerations suggest the expediency of following the operation by the administration of diuretics and heart-tonics. The latter include digitalis and its congeners, which sustain and strengthen the heart-muscle while at the same time they increase pressure in the renal artery, and thus re-enforce any other diuretic remedies. The employment of such agents serves to deplete visceral congestion and to thus enhance the benefit derived from the withdrawal of the fluid which is the original cause of the stasis.

The treatment of purulent effusion should be surgical. Should the nature of the primary infection and symptoms of more or less pronounced septicæmia suggest that the pericarditis is suppurative, the character of the exudate should be determined by exploratory puncture, and if this prove to be pus, it should be evacuted without delay in accordance with proper surgical methods. Lilienthal, of New York, reported before the New York Academy of Medicine a case of suppurative pericarditis, occurring in a lad who was recovering from pneumonia, affecting three lobes. Eighteen ounces of pus were withdrawn at the time of the exploratory puncture, and 40 more at the time the sac was cut down upon and opened. The pus contained numerous pneumococci. The temperature had been irregularly intermittent. Complete recovery followed the operation. Eucaine was used as a local anæsthetic.

Hæmorrhagic effusion is to be managed according to the principles governing the treatment of the sero-fibrinous form, no special indication for treatment being presented by the bloody character of the effusion. No great effect is to be expected, however, from either medical or surgical treatment in those cases in which the affection is associated with a serious blood-state or dyscrasia, and measures should be directed to the removal of these latter.

The subsequent management of a patient convalescing from acute pericarditis consists in such measures as will rapidly restore the general health and heart-tone in the hope that the organ may not be left seriously damaged. We possess no means of either preventing adhesions or promoting the absorption of fibrinous deposits.

CHAPTER II

CHRONIC PERICARDITIS

SYN.: *Adherent Pericardium, Cynechia Pericardii, Concretio Pericardii seu Concretio Cordis*

CHRONIC pericarditis may be divided into two great groups: (1) That in which it involves only the two layers of the sac, *pericarditis interna chronica*, and (2) that in which the process involves both the pericardium and mediastinum, *pericarditis interna et externa chronica*. In both of these forms inflammation results in the formation of fibrous tissue, which binds the parts more or less closely and extensively together. There is still another much rarer form in which the chronic inflammation is associated with serous distention of the sac, and is termed therefore *chronic pericarditis with effusion*.

Adherent pericardium is the term most commonly applied by English writers to the first form, while the second is known as *chronic adhesive (s. fibrous, s. indurative) mediastinopericarditis*. Adherent pericardium has long been recognised by pathologists, but, owing to the difficulty of its diagnosis, escaped clinical recognition, although it is a comparatively frequent post-mortem finding. Out of 86 cases of heart-disease examined after death at St. Mary's Hospital from 1890 to 1893, there were, according to John Broadbent, 31 instances of adherent pericardium.

Although, according to Roberts, Sir Samuel Wilks had frequently called attention both pathologically and clinically to the existence of chronic fibrous thickening within the mediastinum and involving the pericardium, chronic indurative mediastinopericarditis was first systematically described by Kussmaul in 1873. In 1894 Harris, of Manchester, added another valuable contribution to the subject and collected all the published cases. For much of what will be said in the following pages I wish to express acknowledgment to Harris's monograph, and to thankfully acknowledge the stimulus derived therefrom. It has enabled me to give more intelligent and discriminating study to the cases which have come to my notice. I now systematically look for indications of chronic mediastinopericarditis, and discover them many times when otherwise I should probably have overlooked them. Unfortunately, ante-mortem observations of several pronounced cases have been made in which post-mortem corroboration of the diagnosis has been denied. Several instructive and typical instances will be narrated in these pages. I wish also to express my indebtedness to John Broadbent's monograph on this subject, as well as to Friedel Pick's paper, Pericarditic Pseudocirrhosis of the Liver, in which is particularly discussed the effects on the liver and the production of ascites.

Morbid Anatomy.—The morbid anatomical changes found in chronic pericarditis are almost always the result of previous acute inflammation. The more common form is the result of the organization of the fibrinous exudate of an ordinary plastic pericarditis. This process may begin as early as the third or fourth day of the acute inflammation. It is essentially a conservative process, tending to make good the damage wrought by the inflammation. This is brought about by the conversion of the inflammatory exudate into a granulation-tissue, and finally into fibrous cicatricial tissue.

The deeper layers of the exudate are first invaded by newly forming blood-vessels and connective-tissue cells with many leucocytes, which form the granulation-tissue. This gradually grows into and replaces the entire exudate, and in the course of time the development of intercellular substance converts it into the glistening, white cicatrix. If during this process the two layers of the pericardium are in contact, union takes place and the cicatrization produces firm adhesion between the opposing surfaces. These adhesions may be general or local, varying with the extent of the original process, and with the conditions obtaining at the time of organization of the exudate.

When the adhesion is circumscribed it is most frequently found in the parts of the sac where the motion is the least, most frequently, then, at the base of the heart, about the great vessels, less often at the apex or at the borders of the organ. When adhesion does not occur, the formation of scar-tissue produces glistening white areas on the surface of the heart, which show where the previous inflammation existed. These are the so-called milk-spots or *maculæ tendiniæ.*

An intermediate condition between this and the synechia pericardii, or completely adherent pericardium, is that shown when slight circumscribed adhesions have been partially or completely torn apart by the motion of the heart, producing string-like processes of fibrous tissue that may connect the two surfaces, or may, in rare cases, hang loose in the pericardial sac. When the layers are not adherent, in rare cases fluid is found in the sac.

The layers of the pericardium may be adherent with but slight thickening, but it is the rule to find considerable increase in fibrous tissue, especially in the chronic tuberculous form, where it may become extreme.

Calcification may occur, especially following a purulent pericarditis, the pus becoming first inspissated, and then impregnated with lime-salts. Such calcareous plates may be isolated, or may form a complete investment for the heart, resembling a coat of mail (Ziegler). In this case the motion of the heart is permitted by cracks and fissures in the calcareous mass.

Chronic pericarditis is often associated with *chronic endocarditis,* for the reason that they both often have the same rheumatic origin. Moreover, chronic valvular disease seems to predispose to pericardial inflammation.

Secondary to the pericarditis are usually found more or less hypertrophy and dilatation of the heart, with degeneration of the myocardium, which probably are the result of the mechanical hindrance to the heart's action, and also of the visceral changes that are always the result of long-standing circulatory disturbance.

Also associated with the chronic pericarditis may be an indurative mediastinitis. It may exist alone, but its more frequent occurrence in combination with indurative pericarditis renders it appropriate to consider it here. It consists of a more or less extensive hyperplasia of the connective tissue of the mediastinum, which binds together the structures contained therein, and is often associated with adhesion of the two layers of the pericardium. This development of fibrous tissue results either from an extension of a chronic pericarditis through the parietal layer of the pericardium, or from a chronic proliferative inflammation of the mediastinum itself, either alone or associated with pericarditis. Extensive fibrous adhesions may bind the heart-sac inseparably to the diaphragm, or the sac may be united to the anterior chest-wall, to the pleuræ, œsophagus, spinal column, or to all these structures. In some instances the contents of the mediastinum are so matted together by dense fibrous tissue that they cannot be separated without laceration of the organs.

When such extensive adhesions exist they may be found to form but a part of a chronic inflammatory or proliferative process which has led to extensive or general adhesions between the two layers of the pleura, or between the lungs and the diaphragm.

In exceptional cases fibrous adhesions have formed only at the roots of the great vessels, and have led to partial or complete obliteration of the superior vena cava, either alone (Roberts), or in combination with involvement of the main trunk of the pulmonary artery, or the ascending aorta (Kussmaul). At the present writing I have under observation a female patient, in whom, to judge from physical signs, chronic mediastinopericardial adhesions have led to such retraction of the borders of the lungs that the entire anterior surface of the heart is uncovered. The apex-beat is held immovably fixed in the seventh intercostal space, anterior axillary line. The normal excursion movements of the diaphragm in front are entirely abolished, and the respiration is of purely costal type.

The *secondary effects* of this form of the disease are not limited to the heart, as in the simple adherent pericardium. As the disease is usually combined with chronic valvular disease, it is difficult to say how much importance is to be attached to this, and how much to the fixation of the pericardium in the production of the great hypertrophy and dilatation found in these cases. The changes in the lungs of chronic bronchitis and brown induration are due partly to the passive hyperæmia secondary to the cardiac disease, and partly to the retraction and immobility of the lungs incident to the pleuro-pericardial or associated pleuritic adhesions.

Cirrhosis of the liver is generally present, and is largely responsible for the ascites so frequently observed as a terminal event. There may be also thickening and contraction of the capsule of the liver and spleen, and more or less evidence within the abdominal cavity of what appears to have been a general serositis or proliferative inflammation of the serous membranes.

Chronic Pericarditis with Effusion.—This form of pericardial disease is but rarely encountered; when, however, it does exist, it may be considered to have originated in one of two ways: (1) It may start as an acute attack, which, instead of undergoing complete subsidence, suffers repeated exacerbations, and finally merges into a chronic inflammatory process. (2) In consequence of the mildness of the infection the pericarditis assumes a slowly progressive character from the beginning, at no time manifesting a tendency to undergo arrest. In the former class the exudation fluctuates in amount from time to time, according as the intensity of the inflammation abates, and partial absorption occurs, or according as fresh infection occurs the inflammatory process again rekindles. In the second class, chronic from the outset, effusion accumulates slowly, and either remains stationary, after having reached a certain degree, or tends to gradually increase. This form of chronic pericarditis is said to be observed chiefly in elderly or aged individuals, and to be associated with chronic nephritis. Roberts expresses the opinion that such cases can be differentiated only with great difficulty from instances of hydropericardium, and that it is quite possible, indeed, that the chronic pericarditis originated in a simple serous transudation. This, it seems to me, is quite unlikely unless the originally serous effusion becomes infected by pus-cocci, in which event it would likely be transformed into pyopericardium.

Etiology.—Chronic pericarditis is in most instances of either rheumatic or tuberculous origin, the inflammation having been slowly progressive from the start. In other cases an acute process passes into a chronic one, which exhibits no tendency to abatement, but persists for years with repeated exacerbations of the inflammation. This is the case particularly with the form which, starting in the sac, spreads to the mediastinum, and ultimately becomes a chronic fibrous mediastinopericarditis.

In some cases a mediastinitis is first set up and subsequently invades the pericardium. This last form may originate as either a chronic or acute mediastinitis, which is set up by disease of the bronchial or mediastinal glands, malignant tumours, tuberculosis of the lungs, pleura or glands, pneumonia, or by trauma.

The disease is most frequently observed in young adults and in children. Of 22 cases collected by Harris in which autopsy was held, only 2 occurred in persons past thirty, while 9 were under eighteen years of age. Several instances have been reported of its post-mortem discovery in infants. According to Harris, chronic indurative mediastinopericarditis is much more frequent in males than females, 20 out of 25 cases having belonged to the former sex.

Symptoms.—Many cases of adherent pericardium run an absolutely latent course, and are only discovered on the autopsy table. In other cases the symptoms are those of circulatory and respiratory embarrassment, and are attributed to dilatation or to an associated valvular disease. In a third series of cases the synechia pericardii is overlooked owing to the development of ascites and other symptoms of hepatic cirrhosis. For the most part the last-mentioned class of cases belongs to what has been described as chronic adhesive mediastinopericarditis.

The explanation of these clinical differences is not always clear, but probably depends upon several factors. If the pericardial adhesions are of limited extent they may produce no appreciable secondary effects on the heart or circulation, but if they lead to total obliteration of the sac, and particularly if this latter is also bound to some of the surrounding structures, cardiac hypertrophy is likely to result, which, if slight, is not recognised clinically and does not occasion symptoms of embarrassed circulation. If, however, the heart is dilated as well as hypertrophied, it is very apt to be more or less inadequate with resulting respiratory and circulatory symptoms. The enlargement of the organ may be recognised, but not its cause, and the condition is perhaps considered cardiac myasthenia or even chronic myocarditis.

Sir William Broadbent is of the opinion that pericardial adhesions lead most frequently to enlargement of the right heart in consequence of the relative thinness of its wall, while others maintain that the entire heart is hypertrophied. The mode of production of hypertrophy in cases of adherent pericardium is difficult of satisfactory explanation, but is due in some way to the hampering of the heart's work.

In most instances conditions are present which easily account for the cardiac hypertrophy. Chronic valvular disease and adherent pericardium coexist, or the heart is restricted in its function by adhesions between it and surrounding parts (chronic adhesive mediastinopericarditis). In still other instances the effects of a valve lesion are intensified by mediastinopericardial adhesions. In any case the pericarditis either prevents the establishment of adequate compensation or occasions premature loss of such compensation as may have been attained.

When symptoms eventually appear they may be such as are seen in uncomplicated but uncompensated valvular defects; venous stasis, hepatic and other visceral engorgement, œdema, ascites, dyspnœa, and cough with or without expectoration, which may be simply catarrhal or bloody, according to the degree of pulmonary congestion.

In some cases without coexisting valvular disease the earliest symptoms are palpitation, either with or without dyspnœa, called forth by effort or excitement, and occasion much discomfort and alarm. In such the pulse is apt to be habitually rapid, while cardiac impulse is exaggerated in force and extent. In other cases, again, circulatory disturbance is shown by digestive disorders, lasting for many years and attributed to simple dyspepsia or chronic gastritis, but never traced to their proper source because of its obscurity or impossible diagnosis of the pericardial adhesions. In all such cases there is nothing to distinguish them from ordinary instances of cardiac incompetence due to dilatation or mitral disease.

Physical examination usually discloses hepatic enlargement, and if signs of heart-disease are not apparent the condition is thought to be cirrhosis of the liver, either hypertrophic or atrophic, according to the degree of enlargement and density or smoothness of the organ.

In cases of adherent pericardium displaying pronounced engorgement of the liver, I have been impressed by its peculiar obstinacy to treatment. The hepatic congestion is most difficult of reduction by ordinary methods, and displays a striking tendency to recur so soon as treatment is abandoned.

For several years I have had in charge a patient whose mitral valve leaks and whose enormously enlarged heart is apparently completely incased in fibrous tissue that binds down the organ on all sides, so that no amount of rest in bed or digitalis seems to reduce its size in the least. The liver has always been greatly engorged, extending for a long time nearly to the crest of the ileum, and requiring the daily use of saline laxatives to relieve the patient from pain and discomfort. For the past year the organ has been gradually diminishing somewhat in size and increasing in thinness and hardness. The patient has experienced remarkably little dyspnœa on effort, but is greatly annoyed by the pounding and tumultuous action of the heart, this sensation being specially noticeable in the epigastrium. Of late, she has had a great deal of cough, difficult mucous expectoration, and upon several occasions slight hæmoptysis. She has to be extremely careful in her diet, and her urine and menses have become scanty.

Another female patient with pronounced mitral insufficiency has pericardial adhesions that bind down the left side and base of the heart, fixing the apex-beat immovably in place, far to the left and downward, but the border of the right heart is apparently free from adhesions. Whereas the left ventricle never varies in size under any conditions, the right heart, as shown by the area of cardiac dulness, becomes dilated with the greatest ease and rapidity. The liver, which is persistently enlarged, fluctuates somewhat in size in accordance with the state of the right heart, but even when at its smallest always extends from 2 to 3 inches below the inferior costal margin, no matter how vigorous may be the onslaughts upon it by means of Epsom salts. This patient's symptoms are not of the digestive organs, but are those of shortness of breath and a rapid pounding action of the heart and general weakness. The urine remains fairly abundant, and the menses are too profuse and protracted. She is always promptly benefited by absolute rest in bed, a milk diet, cathartics, and digitalis, although this last-named agent never materially slows the heart.

The most interesting class of cases are those whose clinical features closely resemble a case of atrophic cirrhosis of the liver. These cases, usually of chronic fibrous mediastinopericarditis, generally pursue a latent course for many years, and often, even after symptoms have set in, are not recognised as adherent pericardium until they come to autopsy. Not only is there a chronic engorgement of the liver, but there is a perihepatitis with increase of the interstitial connective tissue. In time this fibrous tissue, undergoing contraction, causes a reduction in size and marked increase in the hardness of the liver, which, extending below the costal arch to a variable distance, feels thin, dense, and regular, the notch being intensified, and often one lobe disproportionately larger than the other. It is now, when the organ has shrunken and grown dense, that symptoms begin. The patient's attention is first attracted by an increase in the size and firmness of his abdomen. In some instances icterus accompanies or even precedes this increase of girth.

At length driven to seek medical advice, he is discovered to have slight icterus and ascites, usually without œdema of the lower extremities. The physican examines the heart and urine, detects no heart-disease and discovers no albumin, but perhaps some bile. The case is put down as one of hepatic cirrhosis. The following is an illustrative case:

A man of fifty-five, who had been intensely jaundiced for nearly two years, and in August, 1900, was tapped for ascites, called me in consultation a short time ago. The ascites, which had for a time been reduced by apocynum cannabinum, only to speedily recur, had been again drawn off the morning of the day I saw him. He had had articular rheumatism eighteen years before, but had suffered no shortness of breath or other discomfort since. The thin-bordered, dense, slightly granular-feeling liver extended in the median line nearly to the umbilicus and from one costal arch to the other, being lost beneath the right ribs, just outside the right mamillary line. Owing to the recent paracentesis, the peritoneal cavity was free from fluid, and there was no œdema. The cardiac area was somewhat increased to the right and downward, the sounds were clear and strong and free from murmurs. The rather tapping apex-beat was in the fifth left interspace inside the vertical nipple-line, and there was a distinct though feeble pulsation in the epigastrium. In the fifth and sixth interspaces, between the apex-beat and sternum, and also in the sulcus between the ensiform appendix and adjacent costal cartilages, a systolic retraction could be perceived both by palpation and inspection. Furthermore, when the patient was instructed to take a slow, deep breath, the right external jugular could be seen to bulge out during the inspiratory effort. This distention was also palpable. Pulsus paradoxus could not be determined. I had no hesitation in making a diagnosis of pseudo-atrophic cirrhosis of the liver secondary to an adherent pericardium. This patient died a few weeks later.

One of the most typical cases, and by the way the first of the kind I ever saw, was seen in 1891 with Dr. Christophe. The patient was a male, aged fifty-two, had always enjoyed good health until an attack of the grip in February, 1889, after which his health failed progressively. Six weeks prior to my visit he took to the house with dropsy and ascites. The former yielded to caffeine and digitalis, but the latter persisted until drawn off by tapping the day before I saw him. The patient was in bed, of medium height, considerably emaciated, and complained of dyspnœa, dry cough, anorexia, flatulence, constipation, scanty non-albuminous urine, pain in the hepatic region, and insomnia. The lungs were negative, but on examining the heart the apex-beat was found to be a weak tap in the fifth left interspace on the nipple-line and to be followed by a distinct diastolic rebound or shock, while there was in addition an unmistakable systolic recession of the fifth interspace, from the border of the sternum to a point outside of the apex-impulse. Cardiac dulness extended from the right sternal border to ½ an inch outside the left nipple, and in the mitral area was a harsh systolic murmur that was transmitted to mid-axillary line (Fig. 22). Both heart-sounds were audible, and the second at the apex was followed by a short diastolic murmur. The inferior hepatic border was palpable 2 inches below the costal arch, and was thin, hard, and somewhat irregular. The pulse was slow, tense, and regular, and there was no icterus.

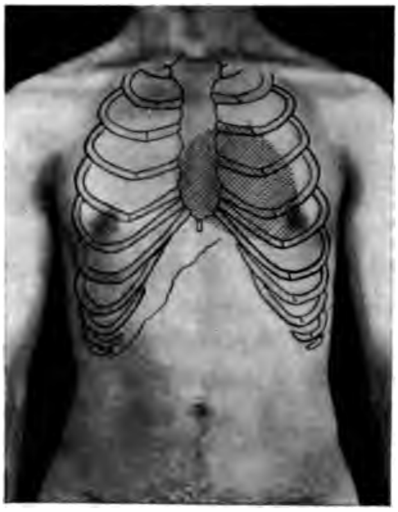


FIG. 22.—SHOWS CARDIAC DULNESS AND LOCATION OF BORDER OF LIVER.

The diagnosis was plainly that of mitral regurgitation and adherent pericardium with secondary cirrhosis of the liver and ascites.

The chronicity of such a case is attested by the fact that after repeated tappings and prolonged confinement to the house this patient again appeared in public, and was accidentally encountered by me in the fall of 1895. He admitted that he was not very well, and that he still had his ascites. He died a few months subsequently. In this interesting case cardiac symptoms did not assert themselves, and the clinical history was essentially that of atrophic cirrhosis of the liver, and would ordinarily pass for such.

The following case is narrated because of its interesting clinical course and instructive pathological findings: Mrs. M., aged forty-five, consulted me in February, 1893, because of an obstinate cough which had developed the November previous and resisted treatment. She gave a history of scarlatina at the age of seven, and of a pain, probably rheumatic, in the right hip almost continually between her tenth and thirteenth years. She stated, also, that at that time she suffered from shortness of breath upon attempting to run or go upstairs, and at such times had an inclination to faint. She thought her pulse had always been irregular, since whenever she had had occasion to require the services of a physican comment was made upon its irregularity. She was married at the age of twenty-one, and two years later gave birth to her only child, both the pregnancy and labour having been uneventful, excepting for a mild "milk leg." Fourteen years subsequently she became a widow. She had considered herself well except for nervousness and attacks of neuralgia. In the fall of 1892 was treated for pain and swelling of right leg, between ankle and knee, and for "fulness and tightness" about the waist. In November had contracted a bronchitis, and since then had not been free from cough, although under treatment for same.

Her only symptoms at the time she consulted me were frequent paroxysmal cough, with scanty mucous expectoration, pain across the chest, in consequence of the cough, and moderate shortness of breath on exertion. Digestion, bowel movements, and micturition seemed normal. She had passed the menopause several months before.

She was 5 feet 1 inch in height and weighed 145 pounds. The pulse was 103, somewhat irregular, not intermittent, small, and weak. The lungs were resonant throughout, and the breath-sounds vesicular; no râles except fine inspiratory crepitus at the extreme right base, close to the spinal column, and at the extreme lower limit of the left lung, from the anterior axillary to the posterior scapular line. These râles were thought to indicate old pleuritic adhesions. The apex-beat was in the fifth left space 2 inches from the sternum, broad, strong, and at times thumping. There was slight epigastric pulsation, and cardiac dulness was increased somewhat to the right.

The pulmonic second sound was accentuated, while the first at the apex was at times split and thumping, at times preceded by a short, rough presystolic murmur. A systolic apex-murmur was not very distinct. No signs of adherent pericardium were noted at that time either because overlooked, or because the chronic mediastinitis did not develop until a year or two subsequently. The lower border of the liver, firm and rounded, was palpable nearly at the level of the umbilicus. There was no œdema.

The diagnosis was made of mitral stenosis in a fair state of compensation, secondary hepatic engorgement, and chronic bronchitis, probably secondary also to the mitral disease.

Under appropriate treatment, directed mainly to relieving stasis in the pulmonic and general venous systems, cough gradually disappeared, and the patient considered herself in fair health during the summer. In May it was noted that the pulse was 100, not intermittent, but slightly irregular in force. The following October, after I had returned from Bad Nauheim, and instituted the balneological treatment of heart-disease in my practice, the patient decided to try a course of baths. For a time they seemed to benefit her, but after about three weeks she said she began to notice increase in the size of her abdomen at its lowest part. I at once examined her, and to my surprise detected unmistakable signs of moderate ascites. The baths were discontinued in the belief that inasmuch as they had not prevented the development of ascites, they would not cause its removal.

From that time onward ascites gradually increased until in June, 1894, paracentesis was performed for the first time. From this time to the date of her death, a period of three years, the fluid was drawn off 32 times, the longest interval between the tappings being seven weeks and the shortest six days. In addition she took elaterin in large doses and various diuretic remedies. Palpation of the liver now showed that it had become thin, very hard, and deeply notched.

About two years before her death she began to suffer from what appeared to be attacks of subacute bronchitis. At such times there was mild continuous pyrexia and the cough was most harassing, often effectually preventing sleep and requiring large doses of codeine phosphate. All known expectorants in various combinations were tried with apparently no more effect than to facilitate expectoration. The only treatment that seemed of material benefit was truly heroic catharsis, since the withdrawal of the ascites seemed only to remove pressure on the diaphragm, but not to lessen the great venous engorgement.

These attacks grew more frequent, the intervals between them shorter, until at last cough became chronic and persisted to the end. During these months I saw her but rarely, as her son, who was a physician, devoted himself to her care. At one of my visits, a year or more before her death, I discovered fine crackling râles all around the base of the right lung, particularly in front, which were brought out distinctly by deep inspiration, and were unchanged by cough. These râles eventually spread so as to be heard nearly to the clavicle, while as time went on similar crepitant sounds became audible more or less extensively at the base of the left lung. They did not seem to have the characters of pleuritic friction, and I was at a loss to explain them. It may here be stated, however, that the autopsy subsequently showed them to have been due to wide-spread pleuritic adhesions.

A year before her death her son first detected systolic recession of the intercostal space, close to the site of the apex-beat, and *pulsus paradoxus*. Bacilli were never discovered in the sputum, and repeated examinations of the urine showed nothing more than the usual changes due to passive congestion. The last months of life were spent in a continual struggle to keep within reasonable limits the ever-present and never-conquerable venous engorgement. Œdema of the lower extremities supervened many weeks before the end, which finally took place in May, 1897, with symptoms closely resembling but not identical with uræmia.

Thanks to the intelligent study of the case by her son, the antemortem diagnosis was made of chronic indurative mediastino-pericarditis. No other signs ever developed than the few mentioned above, *pulsus paradoxus* and a visible systolic recession in immediate proximity to the apex.

The autopsy was made by Dr. W. A. Evans twenty-seven hours after death, and was briefly as follows: The abdomen contained a small amount of fluid, and the omentum was adherent to the abdominal wall above the umbilicus and along the linea alba, adhesions being so firm that they could not be separated without tearing the omentum. The uterus was larger than normal, the right tube very firmly adherent to rectum and posterior part of the uterus, right ovary being normal; left tube also firmly adherent to the left side of the rectum and side of pelvis and posterior wall of the uterus, completely covering the left ovary, which was also normal. There was an exudate upon the anterior surface of the left broad ligament. The liver was adherent to the abdominal wall over both right and left lobes, the parietal layer of the peritonæum being thickened and its visceral layer showing evidence of old inflammation. The organ measured 9 by 5 inches, its right lobe 4 inches vertically, and its left lobe 2 inches in its antero-posterior diameter. The surface of the liver was markedly irregular and divided by scars into large areas, its lower border being so notched that it was practically impossible to make out the lobes. Its capsule was irregularly thickened, presenting the appearance of lace-work. The substance of the organ was firm, cutting with resistance, and the lobules, very irregular in size, stood out prominently, and the connective tissue of the capsule could be traced into the underlying liver substance—in short, it was in a state of advanced Glissonian cirrhosis.

The right kidney, 5¼ inches long and 2½ wide and 2 thick, showed increase in the thickness of its capsule, some parenchymatous degeneration and interstitial overgrowth. The left kidney, 5 by 3 by 1⅝ inches, with capsule firmly adherent in places and thickened, showed other changes the same as in right kidney.

The spleen showed marked thickening and some interstitial splenitis.

The gastro-intestinal tract showed no especial changes, except that the peritoneal covering was thickened.

The appendix was firmly bound down to the right iliac fossa by a solid mass of adhesions behind the cæcum, and was less than 1 inch in length.

The right pleural cavity was the seat of very extensive adhesions, which were most firm anteriorly and more abundant at the apex than at the base. There was considerable fluid in this cavity, and the lung was firmly attached to the diaphragm. The pulmonary pleura was thickened, and on section the surface of the lung showed considerable anæmia, no tuberculous nodules, and no inflammation.

The left lung was adherent at base anteriorly and also posteriorly, adhesions to diaphragm being very solid. At apex was a superficial calcareous mass attached to the visceral pleura, and in other respects the left lung was of the same appearance and condition as the right lung.

The *pericardium* was attached to the pleuræ and chest-wall, and *in situ* felt like a solid bony shield, reaching to the sixth rib and ½ an inch within the left mammary line. Upon removal of the heart it was found that the organ was completely incased by several calcareous plates, which were closely in apposition with yet separated from each other, so that the lines of separation had allowed the heart to undergo contraction and relaxation. These plates of lime united the two pericardial layers firmly. The walls of the several chambers were hypertrophied, particularly the left auricle and right ventricle. With exception of the left ventricle the cavities were all moderately dilated. The heart-muscle had the appearance of brown atrophy. All valves excepting the mitral were healthy, these being thickened, stiffened, adherent along their edges, and projected into the cavity of the ventricle like a cone or funnel. The mitral orifice was moderately thickened with old sclerotic tissue and admitted one finger.

To me it is very interesting and quite remarkable that the patient was never able to give any history of an attack of pericarditis, which the necropsy showed must have been very extensive. It probably occurred at so early a period in childhood, perhaps subsequent to the scarlatina, that it failed to be impressed on her memory, or was not discovered at the time. If it was secondary to the scarlet fever, forty-one years elapsed before it finally led to the patient's death. The post-mortem findings, furthermore, revealed in a striking manner the extent to which chronic proliferative inflammation may involve other structures, notably the liver, and may lead ultimately to the clinical features of hepatic cirrhosis with ascites. The adhesive inflammation of the pleuræ probably occurred at the time when the patient manifested a low grade of fever with cough, and fine, dry crepitus over the front and base of the right lung, and subsequently also of the left. This case also illustrates the chronicity of some of these cases, and the fact that death is the result not so much of cardiac asthenia as of the effect on the system of the associated conditions. That etiological data in such cases are frequently wanting, and that therefore the primary cause of the symptoms may be unsuspected to the end, is shown by the following case:

Mrs. D., a physician's wife, aged forty-six, was first seen by me December 24, 1894, because of increasing symptoms of cardiac disease. Her statements were positive that with the exception of measles and whooping-cough in childhood she had never been ill before the onset of her present trouble. She had been a schoolteacher up to her marriage three years before. Her husband stated that he at first noticed tachycardia shortly after marriage, but no other symptoms had been observed until May, 1894. She then developed dyspnœa on exertion, and occasionally œdema of the lower extremities and face. During the summer of 1894 she took sulphate of strychnine, digitalis, strophanthus "off and on" without apparent benefit, but had recently shown some improvement on iodide of potash and belladonna. Her menses were absent since July; the only symptoms complained of were slight, dry cough, a not very marked breathlessness on exertion, a feeling of weakness, and occasional puffiness of the lower extremities.

She was of medium height and well nourished. Examination discovered signs of fluid in the right pleural cavity, reaching to the lower angle of the scapula, and some fine crackling râles at the extreme posterior left base that grew more abundant after cough.

Otherwise the lungs were negative. The pulse was small, weak, regular, and moderately accelerated. The apex-beat was in the fifth left interspace, just outside the nipple-line, diffused and weak. There was no epigastric pulsation, but at the level of the fourth costal cartilage deep-seated cardiac dulness reached from 1 inch to the right of the sternum to fully midway between the left nipple and the anterior axillary line (Fig. 23). There were no murmurs, but the first sound at the apex was weak and the second at the base reduplicated, the pulmonic second being accentuated. No signs of adherent pericarditis were noticed. The lower hepatic border was palpable at the level of the umbilicus, and was thin and hard. The spleen was not enlarged, and there was no ascites.

The case was considered one of general cardiac dilatation supervening upon a previous hypertrophy of the left ventricle and upon the increased pulmonary blood-pressure occasioned by the fluid in the right pleural cavity. How much of the increase in heart's dulness to the left was attributable to dilatation, and how much to transposition of the organ from pressure by the intrapleural fluid, was left an open question. In the absence of a definite history of pleurisy and of other signs of dropsy, the nature of the liquid in the pleural cavity, whether an exudate or transudate, was a matter of doubt. The liver was evidently cirrhotic. Examination of the urine was negative.

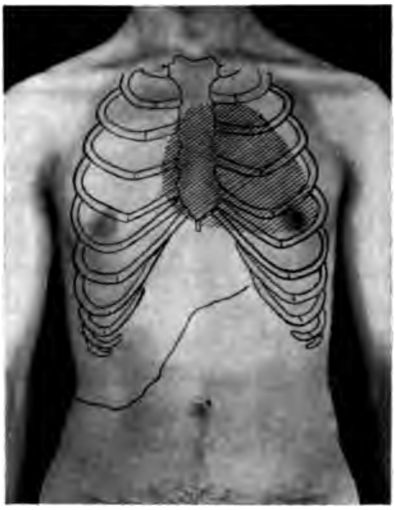


FIG. 23.—SHOWING APEX-BEAT, CARDIAC DULNESS, AND LIVER BORDER IN CASE (p. 114).

In spite of physical rest, digitalis, diuretics, and cathartics, the amount of intrapleural fluid remained stationary for the next three weeks. Paracentesis was then performed, and the fluid rapidly reaccumulating, the operation was repeated within a week. After the second aspiration the fluid mounted to the upper level of the third rib and symptoms of pressure increased. It was then decided to try the efficacy of baths (Bad Nauheim). These were endured so badly, however, that they were discontinued after four days. By February 6th the actual embarrassment had become so pronounced that this fact, together with the failure of paracentesis to permanently reduce the amount of pleural effusion, led the consulting surgeon to advise resection of a rib in the hope that permanent drainage would afford time and opportunity for the heart to regain its former vigour. The proposal having been laid before the patient and her husband, and their consent obtained, the operation was done the next day. Everything seemed to progress favourably for a few days, when suddenly symptoms of pronounced fibrinous pleuritis developed in that side. Temperature rose to 102° F., strength waned rapidly, and the patient died ten days after the operation. I may say here that the infection was subsequently proved to be a pneumococcus one.

The autopsy was made by Dr. Hektoen twenty-four hours after death, and the findings may be briefly stated to have been a totally adherent pericardium, with several plates of lime-salts upon the surface of the ventricles, the largest lamina being about the size of a silver half dollar. The sac was also bound by adhesions to the left pulmonary pleura, but not to the chest-wall. The valves and myocardium seemed normal, there was evidence of recent right-sided pleurisy with collapse of the lung, and acute œdema of the left lung with some fresh diaphragmatic pleurisy on that side.

The liver was cirrhotic, but not atrophied. Kidneys were healthy, and there were evidences of perihepatitis and perisplenitis.

The necropsy seemed to make it evident that the condition within the right pleural cavity had been a hydrothorax, while the pericardial adherence explained why medical treatment had been unavailing. Had this patient lived, and time been afforded for shrinkage of the liver to take place, ascites would undoubtedly have developed, and the case have presented the clinical features of what Pick terms pericarditic pseudocirrhosis of the liver, the same as did the others.

In explanation of ascites in such cases Heideman lays down the four following propositions: (1) In these cases there is chronic inflammation of all the serous membranes. (2) The stagnation occasioned by degeneration of the cardiac muscle leads to ascites, because on account of the chronic peritonitis the peritoneal vessels offer a *locus minoris resistentiæ*. (3) The cirrhotic process in the liver so often observed under these circumstances is occasioned by extension of the inflammatory irritation from the capsule of the liver as well as by the chronic hyperæmia. (4)

By the growth and contraction of this connective tissue in and about the liver the stagnation and transudation in the abdominal cavity are increased.

Course and Termination.—The course of chronic adhesive pericarditis varies much, depending upon the extent of the adhesions and the coexistence or not of other diseases, as chronic valvular lesions. If the process is confined to obliteration of the sac the condition may last for many years, and the patient subsequently die from some independent affection. Usually, however, the same as when mediastinopericarditis leads to extensive union with the surrounding parts, the disease, after having persisted for a long time without symptoms, is likely to bring about those circulatory and respiratory disturbances already described, and so well illustrated by the foregoing cases. If adherent pericardium is associated with valvular disease it affects the latter injuriously, and its more rapid course is inseparably connected with that of the endocardial lesion. In such a case cardiac breakdown is inevitable, and not likely to be long postponed. Nevertheless, even here much depends upon the treatment and upon the patient's intelligent appreciation of his condition. In the case of the young lady whose mitral insufficiency is sadly complicated by adhesion of the left side of the heart and apex to the retracted lung-border and chest-wall, symptoms of right ventricle failure came on only three years after her first attack of rheumatism, and were undoubtedly hastened by the pericardial adhesions (see page 106). It is now nearly eighteen months since her symptoms became alarming, and the fact that her overburdened right ventricle is to-day actually doing better work than a year ago is due to the persistent use of appropriate remedies and to her having learned that so soon as the first signal of danger is perceived she must take to her bed until the right heart recovers its tone.

The manner in which many cases of this affection terminate has already been made apparent. Either the symptoms are those of atrophic cirrhosis of the liver or they are indicative of cardiac insufficiency of mitral disease. Either stasis in the systemic veins and viscera increases until the patient succumbs to general exhaustion, or in another set of cases he is worn out after months or years by the ever-recurring ascites, the heart not evincing special weakness. According to Kussmaul, pulmonary infarcts are particularly frequent in cases of chronic mediastinopericarditis, and these may prove the cause of death.

Physical Signs.—*Inspection.*—The ease and certainty with which adherent pericardium can be recognised clinically depend upon the situation and extent of the adhesions. If the sac is bound down to the heart, but not to surrounding parts, the condition does not of a necessity produce recognisable physical signs, and this fact explains why sinechia pericardii is so often first detected on the post-mortem table. In the cases in which an adherent pericardium is diagnosed there are generally adhesions between the sac and some of the surrounding structures, as the anterior thoracic wall, the pulmonary pleura on either side, and the diaphragm.

Accordingly, it is in cases of chronic indurative mediastinopericarditis that the diagnosis is most easily and frequently made. This is owing to the fact that the existence of adhesions interferes with the change in form and position of the heart normally occasioned by ventricular systole. During this phase in its contractions the heart becomes depressed at its base, and assuming a more rounded shape thrusts its point forward, upward, and towards the left, and thus produces the impulse against the chest-wall known as the apex-beat.

It is evident that if adhesions restrict these movements the heart will of a necessity pull on the part to which it is bound. This pulling action is exerted during ventricular systole, and consequently the most obvious and the most frequently observed sign of adherent pericardium is a *visible systolic recession* of the chest-wall. It may be perceived in various situations, but most commonly in the neighbourhood of the apex-beat. Only a very limited area may be thus drawn inward, but in most instances a systolic sinking takes place in several of the interspaces near the apex and even in the epigastrium, the extent and location of the adhesions determining the extent and position of this sign. It is best observed by placing the patient in a strong light, and then looking at the bared chest from above downward or from one side to the other.

It is well to have the patient suspend respiration for a moment while inspection is being made, that the observer may not be deceived or confused by sinking of the soft parts incident to movements of the diaphragm. Ordinarily, there is but slight difficulty in detecting this systolic indrawing in question. One should be careful, however, not to mistake for a sign of pericardial adhesions the systolic depression that sometimes takes place in the third and fourth interspaces close to the left border of the sternum in cases of great cardiac hypertrophy, and which is due to atmospheric pressure as the base and body of the heart recede from the chest-wall during systole.

As might be expected, it is the yielding soft parts that display systolic retraction most readily, and as it is possible that even near the apex this may be owing to atmospheric pressure, this sign is not pathognomonic. The value of the sign is far greater, therefore, when the ribs and end of the sternum are drawn inward together with the interspaces. I have not observed this, but it is said to sometimes occur.

Sir William Broadbent first described a systolic retraction of the tenth and eleventh interspaces below the inferior angle of the left scapula in cases of adherent pericardium, and hence it is often spoken of as *Broadbent's sign.* It is occasionally perceived on the right side also. It is ascribed to the drawing on the diaphragm of a hypertrophied and powerfully contracting heart, and when present is considered indicative of extensive adhesions between the sac and the diaphragm.

Gibson very justly attaches great importance to *fixation or immobility* of the apex. Normally the heart, and hence its apex, gravitates towards the dependent side whenever the patient assumes either lateral decubitus. When he lies on the left side the point of the heart strikes the chest-wall a couple of inches farther to the left than when he is on his back, while in the right lateral position the impulse is nearly or quite behind the sternum, and hence imperceptible. Consequently in any case in which this mobility of the apex is not observed, it is suggestive, nay indicative, of its fixation by external adhesions. The position of the heart's apex should also become lowered during the inspiratory descent of the diaphragm, striking behind the sixth costal cartilage, or even the sixth interspace. The existence of adhesions may prevent this, and accordingly *fixation of the apex* during the two respiratory acts is likewise a sign of adherent pericardium.

The other phenomena perceived by inspection in some cases are connected with the external jugular veins, and are (1) *inspiratory swelling* of the veins, known as *Kussmaul's sign*, and (2) *diastolic collapse* of the veins, known as *Friedreich's sign*. In my experience these signs are not as frequently met with as is the drawing inward of the interspaces, and I do not recall an instance of diastolic collapse of the veins. Kussmaul's sign is present when pericardial adhesions prevent the dilatation of the right auricle that normally takes place during inspiration. Instead of the inspiratory act exerting an aspirating effect upon the contents of the veins, and thus collapsing them, the opposite effect is produced, and the jugulars become visibly distended. Diastolic collapse does not appear to be limited necessarily to the jugulars, since Broadbent has observed it in the superficial veins on the front of the chest, and says it was due to traction of fibrous bands on the coats of the internal mammary vein uniting this vessel to the pericardium, and causing its sudden dilatation during ventricular relaxation. In the case of the cervical veins their diastolic collapse is probably to be explained by the aspiratory force exerted by the sudden diastolic rebound of the right auricle, pulled upon as it is by adhesions between it and surrounding parts. Two other physical signs that remain to be considered are best perceived by the hand, and are therefore described under palpation.

Palpation.—In some exceptional instances the hand laid over the apex perceives a distinct sudden shock not synchronous with systole, but with diastole. It is spoken of, therefore, as the *diastolic shock or rebound.* It is caused by the pulling of fibrous adhesions which, put on the stretch during systole, pull the heart suddenly back against the chest-wall after systole has ended. Such a rebound can scarcely be occasioned by any other condition than external pericardial adhesions, and therefore by some is considered pathognomonic of the disease under discussion. I have observed it but twice, once in the patient whose case I have reported, and the other time in a man at Cook County Hospital who had, in addition, aortic insufficiency.

The second phenomenon observable by palpation is the *pulsus paradoxus.* Normally the pulse becomes fuller and stronger towards the end of inspiration, smaller and weaker towards the end of expiration. In the paradoxic pulse, on the contrary, the reverse obtains, strong inspiration causing a diminution in the force and volume, it may be even an intermission of the pulse, while towards the close of expiration the pulse regains its usual strength and fulness. This peculiarity may sometimes be perceived in pericarditis with effusion, and therefore, while its presence serves to corroborate other signs of adherent pericardium, it in nowise can be looked upon as pathognomonic. Aside from enabling one to appreciate the two signs just mentioned, palpation is of service in determining the mobility or fixation of the apex and the degree of enlargement and density of the liver—conditions I deem of considerable importance in doubtful cases.

Percussion.—This is of great value in all cases of suspected or known heart-disease. In adherent pericardium cardiac hypertrophy or dilatation is very apt to exist, and hence one should in every suspected case attempt by percussion to ascertain the limits of deep-seated dulness, since in the absence of any other etiological factor to account for enlargement, this condition would point to the likelihood of adhesions.

It is not uncommon in cases of suspected adhesions for the area of absolute cardiac dulness to be increased in all directions, particularly upward and to the left. This may be due to a simple crowding aside of the anterior lung-margins by a hypertrophied heart, yet the borders may be retracted and fixed by pleuropericardial adhesions. In this latter condition the line of demarcation between pulmonary resonance and cardiac dulness is unaffected by respiratory movements. Therefore, percussion is of service in enabling one to determine whether or not the lung-borders are bound down by adhesions.

Auscultation.—For the most part this is of negative value, particularly if the synechia pericardii is not associated with mediastinopericarditis. In the latter condition auscultation sometimes detects fine friction-râles along the margins of the lungs where they join the area of superficial cardiac dulness; and if such parchment-like crackling sounds persist during the cessation of respiratory movements, they furnish strong proof of the existence of pleuropericardial adhesions.

Roberts quotes Perez as authority for the statement that in some instances of chronic mediastinopericarditis a *creaking sound upon the body of the sternum* is audible during up and down movements of the arms. I have tested this in several of my patients, and in two I detected this creaking friction-sound described by Perez. As in these cases other positive signs of adherent pericardium were present, this sign of Perez possessed considerable interest, if not material importance.

Diagnosis.—From the foregoing description of the physical signs it is apparent that in some cases the diagnosis of pericardial adhesions can be made almost at a glance, while in others the most skilful diagnostician may not be able to decide whether the pericardium is adherent or not. The difficulty is found in cases in which the two layers of the sac are united without adhesions to the surrounding parts. In such, one must observe critically the jugular veins and the radial pulse in the hope of detecting some of the anomalies that have been described. The size of the heart should also be mapped out by percussion, and the liver should be examined as to its size, density, and outline, since synechia pericardii may declare itself by no other signs than by its effect on these organs.

In cases of chronic indurative mediastinopericarditis the matter of diagnosis is usually far less difficult. Indeed there may be a conjunction of several physical signs. Thus in one of my patients the apex is firmly fixed far below and to the left of the nipple. There is systolic retraction of the soft parts between the apex and the epigastrium, and of the intercostal spaces below the left scapula. The anterior margins of both lungs are drawn aside and immovable, causing nearly the whole heart to be uncovered, as shown by the great area of superficial dulness. The liver extends nearly to the iliac crest and is hard and deeply notched, while owing to the enormous size of the heart and liver the front of the chest looks distended and smooth, and when the patient stands the abdomen appears disproportionately large and pendulous. Pulsus paradoxus and inspiratory swelling of the cervical veins are also present.

Lastly, one should always be suspicious of an adherent pericardium in every case of rheumatic valvular disease, and if in such a case the liver resembles in thickness and density the organ in atrophic cirrhosis, yet is not so contracted, if ascites develops without apparent cause, or takes place prior to or out of proportion to anasarca, there is good reason to suspect the complication of an adherent pericardium.

The *differential diagnosis* between Laennec's atrophic cirrhosis of the liver and the pericarditic pseudocirrhosis just considered is often the most difficult. Aid may be obtained by attention to the following points: (1) In Laennec's cirrhosis there is often a history of alcoholism, malaria, or syphilis, while in the other form there may be a history of previous pericarditis, a rheumatic attack in childhood, or of some acute illness with præcordial pain and other symptoms suspicious of pericarditis. (2) In Laennec's cirrhosis ascites develops prior to anasarca, whereas in the variety now considered it may sometimes follow more or less œdema of the lower extremities (Pick). (3) In the former there are no signs of heart-disease, while in the latter careful examination usually detects enlargement of the heart either alone or in combination with valvular disease. (4) In the pseudo-atrophic form there may be some of the signs of adherent pericardium.

Finally, before leaving the subject of diagnosis of chronic pericarditis, it is necessary to say a few words concerning the recognition of that rare form in which the chronic inflammation is shown by fluid distention of the sac. In this variety there are apt to be pressure-effects, but if the effusion takes place insidiously such effects may not declare themselves. In such an event the effusion is usually detected accidentally, or if discovered its true nature may not be suspected. Roberts cites a case reported by Samuel West of the existence of a supposed cyst which was repeatedly tapped during life, yet which after death was found to be a chronic pericardial effusion. Unless this condition is observed to have originated acutely its diagnosis must depend upon percussion and auscultation evidences of distention of the sac in accordance with the rules laid down for the diagnosis of acute pericarditis with effusion.

Prognosis.—If pericardial adhesions occur independently of other disease, and if not so firm or extensive as to materially hamper the heart's action, they may in nowise affect life prospects. If, on the contrary, pericardial synechia is complicated by a chronic valve-lesion, the prognosis is unfavourable as to great length of life. If the heart is bound down more or less completely to the surrounding parts, it is only a question of time when serious inadequacy will develop. In some cases the adhesive process is stationary, while in chronic indurative mediastinopericarditis the tendency to subsequent adhesive inflammation of other serous membranes and to the spreading of the adhesive process within the mediastinum furnishes an exceedingly grave outlook for the future. When ascites, anasarca, and other symptoms of the final breakdown appear there is small prospect of a restoration of compensation. Under such conditions the duration of life is likely to be bounded by a few months or even a few weeks. Although, as in the cases narrated, the struggle may be extended over a number of years, the patient is a chronic invalid at the best, and can only with great difficulty postpone the fatal event. The absence of all subjective and objective symptoms furnishes presumptive evidence that the adhesions are not extensive. If, on the contrary, symptoms of engorgement within the lesser and greater circulation are never wholly absent, they afford the basis for unfavourable prognosis. The greater the secondary cardiac hypertrophy and dilatation, particularly in children, in whom chest capacity is small, the slighter the prospect of the long retention of adequate compensation. When the last stage of the journey is reached it is likely to be a short one.

The prognosis of *chronic pericardial effusion* depends upon its etiology and the length of time during which it has existed. It also depends upon its association or not with some other disease, as chronic nephritis, and upon its amenability to treatment.

Treatment.—It goes without saying that we possess no means of breaking up the pericardial adhesions; at the most we can only strive to lessen their ill effects and to prevent an extension of the process. In our endeavour to accomplish the latter, any rheumatic attack or acute illness, no matter how trifling, should be promptly and energetically combated by appropriate means. The patient should be at once confined to the house, and if possible to the bed, in order to relieve the heart of any unnecessary work, and thereby if possible prevent fresh pericardial inflammation or restrain the activity of the process, should the pericardium again become attacked. Salicylates, counter-irritants, or other mild antiphlogistic measures are in order.

The chief aim of management should be to preserve compensatory hypertrophy, and so far as possible to minimize the ill effects produced by the cardiac disorder. In my opinion, the first essential is that the patient be not left in absolute ignorance of his condition, lest he fail to grasp the full importance of the rules laid down for his guidance. Most individuals are greatly alarmed by being told they have heart-disease, and therefore great judgment and tact are required in imparting such information. If the patient has no suspicion of anything being wrong with his heart, and is of a nervous, excitable temperament, he would better be told only a part of the truth. It may be stated that his heart is not strong, and that if he will prevent the development of serious trouble he must obey certain injunctions, the careful observance of which will preserve his health. In other instances the whole truth may be told plainly, but in a manner not calculated to create alarm. Only in this way can we expect our patients, ignorant of physiology and pathology, to avoid harmful efforts, and to correct injurious habits.

In a word, a heart handicapped by extensive adhesions, and perhaps also by serious valve-disease, must not be given more work to perform than it is capable of without strain. Inasmuch as what will be said on this subject in the chapters devoted to the treatment of Valvular Disease in General is applicable to the affection now under consideration, the reader is referred to those chapters for the details of this part of the management.

The injurious secondary effects of adherent pericardium are not limited to the heart, but are also felt by the organs of digestion and elimination. Congestion within the portal system must be diminished from time to time by the administration of a brisk cathartic. The patient, and even the physician, often rest content with the fact that the bowels move regularly every day, and lose sight of the benefit derived in these cases from *periodically unloading the liver*. Nothing is better to this end than a blue pill or a grain or two of calomel, followed the next morning by a glass of some saline aperient water. The patient should remain under the regular, though perhaps not very frequent, supervision of a physician, who, detecting early indications of cardiac strain, may promptly meet the danger by ordering an appropriate heart-tonic. Digitalis and strychnine should not be given as a routine practice, but should be reserved for times of emergency.

As a rule the symptoms pointing to overstrain on the part of the heart can be allayed by regulation of the diet, restricting the amount of work or exercise, and it may be by insisting upon rest in the house for a time. The food should be relatively rich in proteids, moderate in quantity, and taken at regular intervals. If the individual is inclined to corpulence, or suffers from fermentative indigestion, carbohydrates and fats should be allowed sparingly. Unrestrained consumption of fluids is objectionable, since it is a very easy matter for the intake of liquids to greatly exceed the needs of the system and the eliminating power of the excretory organs.

When the breakdown of compensation at length comes, with all its attendant manifestations, the case is to be managed in accordance with the principles governing the treatment of the same condition in any other form of cardiac disease. It has been my experience that one cannot expect or achieve as brilliant results from the employment of digitalis in these cases as in valvular affections unfettered by adhesions. It is not so much a question of whipping on the jaded heart as it is of relieving it of as much of its load as possible. Physical rest must be strictly enforced therefore, and catharsis must be brisk. Digitalis must be given for the purpose of invigorating rather than greatly slowing the heart, and with a view of obtaining its diuretic effect. Diuretin and other diuretic remedies are also in order. It is now that strychnine is of particular service, and to produce its most beneficial effects it should be administered hypodermically. Pain, cough, insomnia, and other distressing symptoms are to be relieved as they arise. One should not hesitate to remove ascites by aspiration whenever it accumulates to the extent of seriously embarrassing the heart and respiratory organs. If the anasarca does not yield to appropriate remedies, it may be drained off by the use of Southey's tubes or by incising the ankles, always under strict aseptic precautions.

CHAPTER III

HYDROPERICARDIUM — HÆMOPERICARDIUM — PNEUMOPERICARDIUM—TUBERCULOSIS OF THE PERICARDIUM—SYPHILIS OF THE PERICARDIUM—CARCINOMA AND SARCOMA OF THE PERICARDIUM

I. HYDROPERICARDIUM

By this term is meant a transudation of serum into the pericardial sac. It is a non-inflammatory process, and the analogue of what takes place under similar conditions in other serous cavities. The presence in the pericardium of 1 or 2 drachms of serum may be regarded as physiological; the condition is pathological only when the transudate reaches such an amount as to constitute a veritable dropsy (hydrops pericardii). Although the condition is the counterpart of transudation into other serous cavities, it does not occur with anything like so great frequency as hydrothorax and ascites.

Morbid Anatomy.—Upon the chest being open the pericardial sac is found more or less distended and fluctuating, the same as in pericarditis with effusion; a great difference is discovered, however, when the sac is opened. Instead of fibrin-masses and other evidences of inflammation, together with a serous exudate, the sac contains a clear, straw-coloured fluid, poor in albumin, and containing very little if any fibrin. Because of its relative deficiency in albumin the specific gravity of the transudate is lower than that of a sero-fibrinous effusion, ranging from 1.008 to 1.015. The pericardial tissues may look more or less œdematous; but aside from this appearance and being filled with serum, the sac presents nothing worthy of note. In addition, there are associated changes in other tissues and organs—such as œdema, depending upon the same cause as the hydropericardium; as chronic diseases of the heart or kidneys, or both, which have served to bring about the serous transudation into the sac. The transudation into the pericardium varies in amount from a few ounces to several pints.

Etiology.—Hydropericardium is a dropsy, and therefore is produced in the same manner and depends upon the same variety of causes as dropsical fluid in other situations. The causes may be divided therefore into general and local. The general include chronic cardiac disease, nephritis, both acute and chronic, and cachexiæ. By local causes are meant those diseases, such as tumours, which, situated within the thorax, exert pressure on neighbouring blood-vessels, and thus bring about stasis in the veins and capillaries of the pericardium. Chronic heart-disease leads to dropsy in the same way, but the stasis within the pericardial vessels is only a part of a general condition.

Symptoms.—These are likely to be overshadowed by those of dropsical accumulation in the pleural cavity and general venous congestion. If by chance hydropericardium exists alone, a very rare event, or forms the leading pathological condition, the symptoms are those resulting from pressure, and consist of the same phenomena of circulatory and respiratory embarrassment as are observed in cases of extensive sero-fibrinous pericarditis. Dyspnœa is more or less marked, and may even amount to orthopnœa; cyanosis and venous congestion are also present, and the pulse is small, feeble, rapid, and it may be irregular. The more rapidly the hydropericardium supervenes the more pronounced the symptoms. As dropsical distention of the sac, when it develops in the course of chronic cardiac or renal disease, is one of the terminal events, it develops so slowly that symptoms are likely to be latent, and therefore escape notice.

Physical Signs.—*Inspection.*—This affords but little if any information, owing to the fact that in most cases distention of the chest has already been produced by associated hydrothorax or the heart and lungs have been crowded upward by ascites. Should some local disease have occasioned the hydropericardium, and the thoracic parietes be sufficiently yielding, there will be more or less præcordial bulging, together with absence of cardiac impulse.

Palpation.—What has been said regarding inspection applies also to palpation. The chief, and perhaps the only thing noted, is *absence of cardiac impulse*, and possibly a sense of increased præcordial resistance. The pulse presents nothing characteristic, since the changes observed in it are also produced by the primary cardiac affection.

Percussion.—This affords us our chief means of diagnosis, the same as in pericardial effusion; for particulars the reader is referred to what has already been said under that head. Owing to the probable association of hydropericardium with hydrothorax, the characteristic shape of præcordial dulness is likely to be modified by and merge into that of the latter affection. Under such circumstances, it would only be at the upper part of the sternum that percussion might be of any special value as regards the detection of the hydropericardium. If the area of cardiac flatness extends high up towards the suprasternal notch, with a bluntly rounded apex, well above the area of dulness due to the hydrothorax, this fact might, theoretically at least, be of aid in determining the existence of transudation into the pericardium.

Auscultation.—Owing to the intervention of fluid between the heart and the chest-wall, cardiac *sounds are feeble and distant,* and they may indeed be almost inaudible. If murmurs, due to some pre-existing valvular disease, are also present, these are likewise enfeebled.

Diagnosis.—From the foregoing considerations it is evident that the *diagnosis* of hydropericardium is not only difficult, but may be actually impossible.

In those extremely rare cases of pericardial dropsy due to local causes the diagnosis is governed by the same principles as in massive pericardial exudation.

The *differential diagnosis* between these two conditions is to be made by the history, symptoms, associated diseases, and presence or absence of pericardial friction. In effusion there is history of rheumatism or some acute infectious disease, of pyrexia, præcordial pain, palpitation, etc. Even in distention of the sac pericardial friction-sounds may be retained. In hydropericardium, on the other hand, there is history or evidence of some chronic valvular or renal disease, and all symptoms of acute inflammation are wanting, and there is no pericardial rub.

Prognosis.—This is unfavourable, both because of the nature of the primary disorder to which the hydropericardium is secondary, and because the distention of the sac is likely to hasten cardiac failure. The prognosis is also influenced by the amenability to treatment of the cause of the dropsy.

Treatment.—This resolves itself essentially into the treatment of the primary disorder, since with the removal of the general dropsy the fluid within the pericardium is absorbed. Unless the symptoms be exceedingly threatening, surgical treatment, if not actually unwise, affords only a very temporary relief. In other words, the treatment of hydropericardium is unavailing unless its cause can be removed. The management of dropsy when associated with chronic cardiac disease, will be found fully narrated in subsequent chapters.

II. HÆMOPERICARDIUM

By this term is not meant hæmorrhagic pericarditis, but an extravasation of blood into the pericardium independent of any inflammatory process. It is fortunately a rather rare condition, and yet occurs many more times than it is recognised. It requires but very brief consideration.

Morbid Anatomy.—As with serous transudation or effusion, the escape of blood into the pericardium causes a distention of the sac proportionate to the amount of the extravasated blood. If the hæmorrhage takes place rapidly the amount discovered post mortem is usually not large, because it has speedily occasioned the death of the patient. If, on the other hand, it takes place slowly, the sac may be greatly distended. The blood may be wholly fluid or have undergone more or less coagulation. After the evacuation of the pericardial contents, careful scrutiny discovers evidence of some one of the causes of the hæmorrhage.

Etiology.—Blood may be effused into the pericardium in consequence of external injury, as by gunshot or stab wounds, laceration of the sac by the sharp edge of a fractured rib, etc. It also follows rupture of the heart-muscle, the bursting of an aortic aneurysm, or in rare instances, of one of the coronary arteries. It is stated that sacculated aneurysms of the ascending arch frequently rupture into the pericardium. Of 953 cases of aortic aneurysm analyzed by Hare and Holder, death took place from rupture 289 times, and of these, 75 cases ruptured into the pericardium. Rupture of the heart occurs from degeneration of the myocardium, and is fortunately a comparatively infrequent event.

Laceration of the heart-wall has occasionally been observed to follow a crushing injury to the chest.

Symptoms.—As would naturally be expected, hæmorrhage into the pericardial sac occasions the very gravest symptoms. If this takes place slowly through a minute slip in the wall of the heart or aorta, symptoms come on gradually, and are those of acute anæmia, together with slowly induced and progressive heart-failure. These are a sense of præcordial distress, anxiety, weakness and prostration, dyspnœa, pallor with cyanosis, a weak, rapid, perhaps irregular, pulse, coldness of the extremities, and clammy perspiration. Death takes place within a few hours, or perhaps a day or two.

Should the hæmorrhage be free, and the sac become rapidly distended, the symptoms are those of sudden and profound shock, the patient passing quickly into a state of collapse, and dying in a few minutes. If the rupture does not occasion appreciable pain there may be nothing in the symptoms to direct attention to the pericardium. In most instances the course is rapid, leading to a speedily fatal termination.

Physical Signs.—These are the signs of fluid distention of the sac, and hence do not require repetition. In the majority of instances death is too rapidly induced or the distention of the pericardium too small to occasion appreciable physical signs.

Diagnosis.—If the life of the patient is sufficiently prolonged, and if the sac is sufficiently filled, it is possible for the true nature of the difficulty to be recognised by examination of the præcordium. If the presence of fluid in the pericardium is made out, the history of its sudden appearance and the symptoms of shock and collapse will probably enable one to surmise at least the true nature of the malady and to differentiate it from hydropericardium. Diagnosis may also be facilitated by history of some antecedent affection as aneurysm, likely to lead to hæmorrhage.

Prognosis.—If hæmopericardium results from trauma, the prognosis depends upon whether or not the injury is amenable to surgical treatment. In cases due to aortic or cardiac rupture the prognosis is absolutely unfavourable, and death is the inevitable, it may be the immediate, result.

Treatment.—This in traumatic cases is surgical, and is best left to text-books on surgery. In the other class of cases there is no treatment, except possibly in those rare instances of traumatic laceration of the heart, when the surgeon should promptly lay open the sac, evacute the blood, in the hope of discovering the source of the hæmorrhage, and of being able to repair the injury by suturing the heart-muscle.

Medicinal treatment is limited to stimulation of the heart and an attempt to support the powers of life. In most instances the physician arrives on the scene too late to do more than witness the death-struggle or sign the death-certificate.

III. PNEUMOPERICARDIUM

This is so extremely rare an affection that but few have been so fortunate as to observe an instance of the kind. By this term is meant a collection of air or gas within the pericardial sac, and hence it is the counterpart of the condition known as pneumothorax.

Morbid Anatomy.—Pneumopericardium is usually associated with collection of fluid, most commonly of pus, within the sac. The amount of contained air or gas is variable, but is sufficient, together with the exudation, to occasion great distention. If the gas is not absorbed, and the pericardium be opened post mortem, the gas escapes with a hissing noise and often possesses a fœtid odour. In some instances its avenue of entrance can be easily ascertained, while in others there is no discoverable opening into the pericardium, either because, if such have existed, it has become closed, or because the gas has been generated *in loco.* There are usually present also evidences of acute pericarditis.

Etiology.—Pneumopericardium may be produced in any one of three ways: (1) Perforation of the sac from without may allow of the entrance of atmospheric air; (2) communication may be established between the sac and some portion of the digestive tract, thus permitting the ingress of the gases normally existing in the latter; or (3) gas may be generated within the pericardium without solution of its continuity. The entrance of atmospheric air into the sac usually takes place through a perforating wound, as from a bullet or some stabbing instrument. In rare instances air may enter the pericardium through the lung in consequence of laceration by the sharp edge of the fractured sternum or rib, or by rupture of a pulmonary cavity situated in immediate contiguity to the sac.

When a communication is established between the pericardium and œsophagus, air is forced into the former with each act of swallowing. Walshe has narrated an interesting case of perforation of the pericardium, and resulting pneumopericardium, during an attempt by a juggler to swallow a short sword.

When gas is admitted to the sac from some one of the hollow viscera it is usually in consequence of the extension of a previously existing ulcerative process. The most frequent communication formed in this way is with the stomach, by reason of an ulcer, when situated on its posterior wall. Of 28 cases of perforation of the diaphragm by gastric ulcers collected by Ludwig Pick, only 10 were cases in which the ulceration had perforated the pericardium. Collingwood Fenwick records a very interesting case which occurred in his practice, in which a gastric ulcer had perforated the pericardium with an immediately fatal result, and yet no previous symptoms had occurred to point to the presence of the gastric ulcer. In 4 of the 10 cases collected by Pick the two surfaces of the pericardium had become adherent before the ulceration had perforated, so that the ulcerative process involved the substance of the heart itself. Ulceration into the sac may also take place from the œsophagus, but, as already stated, atmospheric air is then admitted, instead of stomach or intestinal gases.

In the minds of some, the spontaneous development of pneumopericardium is a matter of doubt. Gibson is of the opinion that in some of the cases supposed to be of this origin the pneumopericardium was in reality the result of an opening into the sac, which, however, became so quickly and perfectly closed that no trace of such opening could be discovered. Nevertheless, the discovery of gas-forming bacilli renders intelligible and possible the spontaneous development of pneumopericardium, and may explain some cases that would be difficult or impossible to account for on any other hypothesis. This mode of production is exceedingly infrequent, to say the least. Such a pneumopericardium is preceded or accompanied by acute suppurative or hæmorrhagic pericarditis.

Symptoms.—Subjective manifestations are essentially those of sudden distention of the sac from any other cause. They are symptoms of pressure; but inasmuch as the entrance of gas takes place suddenly, symptoms develop rapidly and are extreme. In some instances there are symptoms of shock; a weak, irregular pulse, a pale, anxious countenance, the skin covered with cold sweat, which, together with orthopnœa, produces a picture of mortal agony. The physician's attention is at once directed to the heart, the examination of which reveals a most singular group of objective symptoms.

Physical Signs.—*Inspection and Palpation.*—These afford evidence of pericardial distention but not of the nature of the disease, and are of minor importance since the diagnosis is readily established by other means of exploration at our disposal.

Percussion.—The phenomena perceived by percussion are unique; instead of cardiac dulness, encroached upon and surrounded by pulmonary resonance, the præcordium is found to be tympanitic, either throughout or at its upper portion. If the pericardium contains fluid, as well as air or gas, there is dulness over the dependent part and high-pitched tympany above. Gas being lighter than the exudate, change in the patient's position from the recumbent to the upright, and from side to side, causes the gas to move about, so as to be always above the level of the fluid; hence there is change in the location of dulness and tympany according to the posture of the patient. In the erect position dulness occupies the bottom and tympany the apex of the sac. If the patient lies on his left side the fluid gravitates in that direction, with corresponding dulness surmounted by tympany; and, on the other hand, the assumption of the right lateral decubitus causes a corresponding alteration in the relative position of the gas and liquid, with resulting transposition of tympany and dulness. Moreover, the larger the amount of exudate the smaller the space allotted to the gas, and hence the higher the pitch of the tympanitic note. Stokes claimed in one case to have detected "cracked-pot" resonance.

Auscultation.—Perhaps the most striking features are the peculiar sounds observed on auscultation. The movements of the heart cause an agitation of the gas and liquid contents of the sac, and hence a true *succussion-sound* or *splashing.* This is variously described as churning, splashing, or like that produced by a water-wheel. These are sometimes accompanied by that musical sound known as the metallic tinkle, likened to the dropping of water. This pericardial splashing is of precisely the same character as the hypocratic succussion-sound elicited by shaking a patient with pneumohydrothorax. In some instances these metallic sounds are audible at a distance from the patient's chest.

Diagnosis.—This combination of a clear ringing tympanitic percussion-note with splashing in the cardiac area is so unique that an erroneous diagnosis can scarcely be made. It seems to me, therefore, far afield to discuss the differential diagnosis between this affection and dilatation of the stomach or pulmonary vomica in immediate proximity to the heart, which are conditions said by some authors to render a mistake in diagnosis possible. Finally, the confounding of this disease with the presence of air and fluid within the pleural cavity is scarcely likely, if one will bear in mind that in pneumothorax the succussion-sound is only obtained when the patient's body is agitated, while in the affection under discussion the peculiar sound is present even when the patient is at rest.

Prognosis.—This is always serious, yet in traumatic cases there is hope of cure through surgical interference, while the same may be said regarding cases associated with purulent pericarditis. Should gas gain entrance to the pericardium, and be not followed by infection of the sac and inflammation, there is a possibility of its ultimate absorption. Moreover, the prognosis depends upon the suddenness of the formation of pneumopericardium. If this is sufficiently rapid to occasion symptoms of shock, there is strong likelihood that the patient will succumb. If, on the other hand, the condition develops slowly, symptoms may not be very urgent, and time may be allowed for surgical intervention.

Treatment.—It goes without saying, that in most cases if surgical skill cannot remove the cause, other treatment, no matter how energetic, will be found unavailing. The same principles govern the therapeutic management as in other forms of pericardial disease. Supporting and stimulating measures are aways indicated, and may even enable the patient to rally from the initial shock. Pain and distress should be relieved by a dose of morphine administered hypodermically. Heat should be applied to the extremities. Camphor, ammonia, ether, and brandy are useful stimulants, and should be given freely. The physician should not forget the great value of strychnine and digitalis in supporting the heart; the former should be administered under the skin.

IV. TUBERCULOSIS OF THE PERICARDIUM

Morbid Anatomy.—Tuberculosis produces in the pericardium all of the characteristic lesions to which it may give rise in other regions of the body. The process may be acute or chronic in course, and either exudative, productive, or destructive in nature.

The acute process is not often to be distinguished from an ordinary acute pericarditis except by microscopic and bacteriological methods. The exudate may be fibrinous, sero-fibrinous, or purulent, but tuberculous pericarditis shares with the inflammation of malignant disease the distinction of being the most frequent cause of hæmorrhagic exudation in the pericardium. Tubercles may not be demonstrable post mortem, and when found are often exceedingly small, even microscopic. They are usually found on the parietal layer of the sac, owing to the frequency with which the infection extends from neighbouring viscera. The miliary tubercles may be covered by the fibrinous exudate, and are then to be discovered by detaching the fibrin. They may, however, be easily seen, and when collected in groups give rise to areas of caseation. These caseous areas may invade the myocardium, and in one instance a cheesy mass had perforated the wall of an auricle and projected into its cavity, being covered by a thrombus where it was in contact with the blood.

Again, when the production of granulation tissue is the predominating feature of the process, the two layers of the pericardium are bound together by a bluish translucent mass of new-formed tissue. This of course becomes white as it grows older, and the condition of adherent pericardium is produced. Only rarely is the caseous mass calcified after the cessation of the active process—calcification being more common in the inspissated remains of purulent exudates.

Acute pericarditis is probably tuberculous in a larger proportion of cases than has been supposed, and indeed cannot be considered a rare condition. Of 1,048 autospies on adults dead from all causes, Wells found this condition in 10 cases, or nearly 1 per cent; and since in 128 cases the pericardium was actively involved, the 10 cases amount to about 8 per cent. Osler reports 7 per cent from his series of cases.

Chronic tuberculous pericarditis may follow an acute attack, and in this case the post-mortem findings are those of an ordinary chronic pericarditis, with the addition at times of grayish tubercles, or of areas undergoing caseous degeneration. It is the exception, however, rather than the rule, to find distinct evidences of tuberculous origin, even in cases in which the clinical history almost conclusively proves this. Indeed, in spite of the large number of cases of acute pericarditis that are tubercular, and which pass on into the chronic form, it is very exceptional to discover any conclusive post-mortem evidence of tuberculosis in cases of chronic pericarditis. The findings include thickening of the membrane and more or less complete adhesions of the two layers, the translucent bluish granulation tissue of the acute stage having been replaced by firm, white cicatricial tissue.

Pericardial tuberculosis may be chronic from the outset, in which event the lesions are more apt to be of a distinctly tuberculous nature, tubercles and caseating areas being common.

Etiology.—Authors distinguish a primary as well as a secondary form of pericardial tuberculosis. According to Osler, the primary form may be "associated only with caseation of the bronchial or, particularly, the anterior mediastinal glands." In other cases there are no such associated lesions, and in these the tuberculous affection of the pericardium is impossible to explain.

The secondary form is the one generally encountered, and depends upon previously existing tuberculous disease elsewhere in the body. This may be caries of a vertebra, a rib, or the sternum, caseous bronchial or mediastinal glands, tuberculosis of the lung, pleura, or retroperitoneal lymph-glands, or tuberculous peritonitis. Occasionally miliary tubercles within the pericardium are a part of a general miliary infection.

This form of pericardial disease is most common between the ages of fifteen and thirty, yet has been seen in individuals at either extreme of life. Osler met with a case in a child of five, Duckworth in an infant of only five months, and Lajard in a woman of eighty-eight. A patient of mine who died of pulmonary consumption, with acquired dextrocardia, and in whose adherent pericardium tubercles existed, was a young man of eighteen. The disease affects both sexes, but for some strange reason appears to be rather more common in males. Other predisposing causes are all those conditions that render an individual susceptible to this form of infection.

Symptoms.—In most cases the disease is wholly latent, and is only discovered on the autopsy table. This is due to the fact that the disease is generally subacute or chronic, and arises insidiously. If it gives rise to acute inflammation with effusion the symptoms are those of acute pericarditis from other causes—pain, palpitation, fever, friction-sounds, and, upon distention of the sac, the pressure-effects already considered. Even an exudative pericarditis of this origin may in some instances pursue a chronic course.

Physical Signs.—Objective manifestations of the disease are wanting unless the affection is declared as an acute process. When such is the case there are the fremitus, pericardial friction-sound, and, with filling of the sac by exudation, the evidence of fluid distention—i. e., triangular area of dulness and disappearance of the cardiac impulse, etc.; in short, the signs already described in the chapter on Acute Pericarditis.

Diagnosis.—Owing to the insidious onset and latent nature of this disease it is rarely diagnosticated during life. If in the course of pulmonary tuberculosis or of pleuritis in a tuberculous subject the physical signs of pericardial involvement should make their appearance, one might with confidence make the diagnosis of pericardial tuberculosis; but without such favouring conditions it is not at all likely that the disease would be discovered.

Prognosis.—This is not always serious so far as it affects the duration of life, yet it undoubtedly contributes its share to the unfavourable termination of the general tuberculous disease. Its remote effects are an adherent pericardium and cardiac insufficiency. The appearance of a tuberculous pericardial effusion in the late stages of pulmonary tuberculosis, the patient being already cachectic, would undoubtedly hasten the fatal issue.

Treatment.—This is to be conducted on the lines already laid down for the management of other forms of acute pericarditis.

V. SYPHILIS OF THE PERICARDIUM

Invasion of the pericardium by syphilis is so rare an affection that most text-books on diseases of the heart either do not consider it at all or give it the very briefest possible mention. Eichhorst, for example, simply states that gummata in this situation have been described by Lancereaux and Orth. In Allbutt's System of Medicine I fail to find any mention of syphilis under diseases of the pericardium, and the same may be said of Hayden and Walshe in their classical works on the heart. Gibson, whose remarks on this subject are more voluminous, devotes but a single page to it, and would seem to have been largely indebted to Mracek's paper, which has likewise furnished the inspiration for the following brief consideration:

Morbid Anatomy.—Syphilis of the pericardium is always a very rare affection, and is almost never met with unassociated with syphilitic changes in the heart-muscle. When present, the disease is limited to the visceral layer and manifests itself either as gummata or circumscribed thickenings. Although cases have been described as syphilitic pericarditis with sero-fibrinous exudation, Mracek is of the opinion that their syphilitic nature is open to doubt. Of the two forms in which pericardial syphilis declares itself, fibrinous thickening is much the more common. At the time Mracek's monograph appeared only 3 authenticated cases of gumma within the pericardium had been described—one each by Lancereaux, Orth, and Mracek.

The portions of the epicardium that appear thickened and fibrous are usually found to overlie and be intimately connected with areas of pronounced myocardial fibrosis or a gumma situated within the heart-muscle. The development of connective tissue usually begins in the immediate neighbourhood of the blood-vessels, and then extends more or less widely into the surrounding parts.

In the second case of Mracek's series, in which the epicardium was thickened and elevated in a circumscribed zone, immediately overlying a small gumma, the microscope revealed signs of recent inflammation of the adipose tissue. This tissue was thickly infiltrated with cells, particularly in those parts next to the muscle-substance and around the borders of the gummy tumour. Immediately above the gumma there was, in addition to cellular infiltration, a pronounced hyperæmia of the veins and capillaries. On the overlying surface of the epicardium the fibrous tissue was old and firm.

In the course of time the blood-vessels supplying the newly formed connective tissue undergo obliteration, and the latter becomes transformed into firm cicatricial tissue. Thickening of the serous membrane is not necessarily associated with a deposit of fibrin, and consequently pericardial adhesions are not always observed. In some instances, however, the two layers are found loosely united in the areas in which the connective tissue has undergone hyperplasia. Total obliteration of the sac is almost never encountered.

Syphilitic pericarditis is a very chronic process, much more so than is tuberculous pericarditis; and Mracek affirms that in those cases in which it is difficult to determine whether the process is tuberculous or syphilitic, the presence of a sero-fibrinous or hæmorrhagic exudation tells in favour of its tuberculous origin. In cases of exudative pericarditis syphilis is the last thing to be thought of. In some instances the sac may be found to contain a small amount of clear serum, but when present this is a transudation due to compression of the blood-vessels by the products of syphilitic disease. Very rarely hæmopericardium may also be produced, as in one case by a rupture of a small cardiac aneurysm, itself the result of syphilitic fibrous myocarditis.

Etiology.—Syphilitic disease in this situation is a late manifestation of the infection. There are no known factors which determine its invasion of the pericardial sac, but, as it is a constitutional disease, it is only singular that it is not more frequently present in this location.

Symptoms.—Syphilis of the pericardium either occasions no symptoms, or these are obscured by those of syphilitic disease in other parts of the body or in the heart-muscle. Inasmuch as pericardial changes of this nature are almost always found in connection with luetic disease of the myocardium or endocardium, it is impossible to say how much, if any, of the symptomatology is to be attributed to the pericardial disease. It is highly probable, however, that the chief rôle in this respect is played by the myocardial degeneration or the sclerotic endocarditis, as the case may be. The cardiac manifestations will be fully described in the chapter on Heart Syphilis.

Physical Signs.—Except in the rare cases in which the pericardial changes lead to the development of a friction-sound or to dropsical distention of the sac, there are no distinctive objective signs of the local disease.

Diagnosis.—Even in a luetic patient with distinct cardiac symptoms, they are far more likely to be due to syphilis of the myocardium than of the pericardium, and hence one should be very guarded in making the diagnosis of the latter condition. The *intra-vitam* recognition of pericardial syphilis is on the whole, therefore, very unlikely.

Prognosis.—*Per se* pericardial syphilis cannot be regarded as a dangerous affection; the adhesions it induces are usually so circumscribed and loose that they probably exert little if any injurious influence in the way of cardiac hypertrophy and dilatation. In general it may be stated that the prognosis is that of syphilitic disease of the heart-muscle, which, as experience shows, is very amenable to proper management.

Treatment.—This consists of the vigorous employment of mercury and the iodides, and need not here be discussed.

VI. CARCINOMA AND SARCOMA OF THE PERICARDIUM

Malignant disease, like syphilis, attacks the pericardium with such infrequency as to merit but brief consideration.

Morbid Anatomy.—Owing to the extreme rarity of primary tumours of the sac, but little can be said concerning them. Williams and Miller have reported a case of sarcoma of the pericardium in a boy of thirteen. The tumour was a diffuse, small-celled sarcoma of the parietal layer, which had produced uniform thickening, but had not invaded the epicardium. There was no discoverable involvement of the lymph-nodes in any other part of the body, and for this and other reasons the authors concluded that the growth had originated in the lymphatic structures of the sac itself.

Of secondary tumours, those most commonly invading the sac are lymphosarcoma from the mediastinal nodes, and carcinoma from the stomach or œsophagus. The new growth may uniformly infiltrate the parietal layer of the sac, or single nodules may project into its interior. There is always more or less fluid in the sac, either of inflammatory or of dropsical nature. The inflammation due to cancerous disease of the pericardium is particularly apt to produce a hæmorrhagic exudate—being in this regard like the tuberculous disease.

Etiology.—Primary malignant disease of the pericardium is so rare that, according to Gibson, the only authentic case on record was the one observed by Koester. Sir William Broadbent has, however, reported an instance of sarcoma, which was thought to be primary, and I have mentioned above the case reported by Williams and Miller. In the vast majority of cases this affection of the sac is secondary to new growths in other situations, as in the œsophagus, lungs, pleura, mediastinal glands, liver, etc.

Symptoms.—As a rule this disease of the pericardium is latent or the clinical picture is that of the primary tumour.

Physical Signs.—So far as known, there are no distinctive physical signs of malignant invasion of the pericardium. If such are produced, they are those of secondary inflammation or of dropsical distention of the sac, and require no repetition.

Diagnosis.—This is rarely if ever possible, and would naturally depend on objective manifestations of pericardial disease, which, as just stated, are very uncertain.

Prognosis and Treatment.—The former is hopeless, since the disease is not amenable to surgical interference, and the latter must be confined to measures calculated to relieve suffering and promote euthanasia.

SECTION II

DISEASES OF THE ENDOCARDIUM

CHAPTER IV

ACUTE ENDOCARDITIS

This is an inflammation of the lining membrane of the heart, which it has long been customary to divide into two forms, for reasons apparent in the various adjectives applied to them. Thus one is called simple or benign, because it does not often destroy life directly, but permits the patient to recover, although with valvular lesion. The terms vegetative and verrucose are also applied to this variety, particularly by the Germans, on account of the nature of the inflammatory changes induced. Simple and benign refer to its clinical manifestations, verrucose and vegetative to its anatomical characters.

The other form, fortunately much less frequent than the preceding, is spoken of as malignant, to designate its usually fatal ending; and infectious or infective, in allusion to certain etiological and clinical characteristics. Its anatomical features, on the other hand, are shown by its other names—ulcerative, diphtheritic, mycotic. Diphtheritic was applied to it by Virchow, and mycotic by Winge, who was the first to describe microbes in the valves.

In conformity with the plan of this work, which is to designate diseases by their most familiar and generally employed names, these two affections will be spoken of as *acute simple* and *acute ulcerative* endocarditis. In accordance with custom, moreover, they will be considered as distinct clinical entities, although I am not unmindful of the fact that a sharp dividing line cannot always be drawn between them either clinically or anatomically.

The endocardium may become inflamed during fœtal as well as extra-uterine life; but the two halves of the heart are affected with different degrees of frequency during these two periods of existence. After birth it is the lining membrane of the left side that is generally attacked by inflammation, as is so well shown by Sperling's oft-cited statistics of 300 cases at the Berlin Pathological Institute. Of these, the left side alone was found affected 268 times, right heart alone 31 times, both together 29 times. Of the cases affecting the left side, the mitral valves were involved 255 times, the aortic but 129 times.

Morbid Anatomy.—The endocardium is the lining membrane of the heart, and is continuous with the intima of the blood-vessels through the various openings in the auricles and ventricles. It consists of two laminæ—a fibrous, very thin in most portions, and an endothelial, the latter consisting of a single layer of flattened cells, which are in contact with the blood.

The valves of the heart are folds of the endocardium, the fibrous layer being increased to give them greater strength. The valves contain no muscular tissue, and are avascular, with exception of the attached margins of the mitral and tricuspid leaflets, which contain a few very small vessels. These are the only portions of the endocardium that contain blood-vessels, as the mural endocardium, as well as the remaining portions of the valves, derives its nutriment from the blood passing over it. Lymphatics are, however, numerous.

Anatomically, it is exceedingly difficult to draw any sharp distinction between the benign and malignant forms, as all intermediate grades are found and the differences seem to be only those of intensity of the process. These differences are doubtless dependent on infection by different organisms, of which a large number have been described by different investigators.

In the *simple form* the first change visible to the unaided eye is a cloudiness or opacity of the membrane. This is probably in all cases preceded by the lodgment of micro-organisms on the surface, which had been rendered vulnerable by some previous injury. Wyssokowitch, Prudden, and others have shown by animal experiments that cultures of pathogenic bacteria, injected into the circulation, produce the lesions of endocarditis only when the endocardium has been previously injured, as by passing a probe down the carotid artery or jugular vein. This probably explains the fact that the lesions are most often found on the valvular endocardium, as these portions are most liable to injury. In intra-uterine life endocarditis is most common in the right heart, and more often on the tricuspid than on the pulmonary valves.

In extra-uterine life the lesions are most common on the mitral valve, next on the aortic, and only very rarely on the valves of the right side. Furthermore, the lesions are usually found, not on the free margins of the valve-cusps, but along a line corresponding to the point of maximum contact when the valves close (Fig. 24). In the case of the auriculo-ventricular valves this is on the auricular surface, while on the semilunar valves it is on the ventricular surface. From these facts it is evident that the work that the valve has to do and the strain to which it is subjected are factors in the determination of the location of the process.

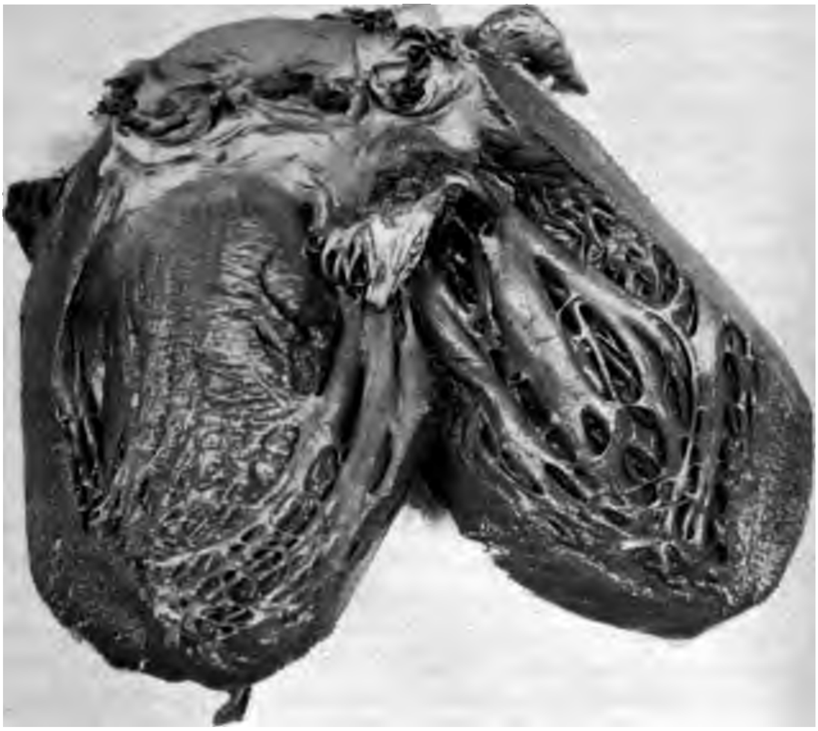


FIG. 24.—VERRUCOSE ENDOCARDITIS OF AORTIC AND MITRAL VALVES.
Specimen in collection of Dr. Gustav Fütterer.

Following the appearance of cloudiness, the membrane becomes thickened and œdematous, while the straining and pounding to which the segments are subjected are very apt to produce erosions or lacerations. These naturally occur at the points weakened by the invasion of micro-organisms, and if the eroded surface is not at once covered by the deposit of fibrin from the blood, a considerable loss of substance may take place. This is far more common, however, in the malignant form, although it has been observed in simple endocarditis complicating rheumatism. More commonly the eroded surface, necrotic from the action of bacteria, is at once covered by a deposit of fibrin from the blood. This fibrin forms a firm warty mass of a yellowish or reddish colour, which rises above the surface of the membrane, and hence has received the name

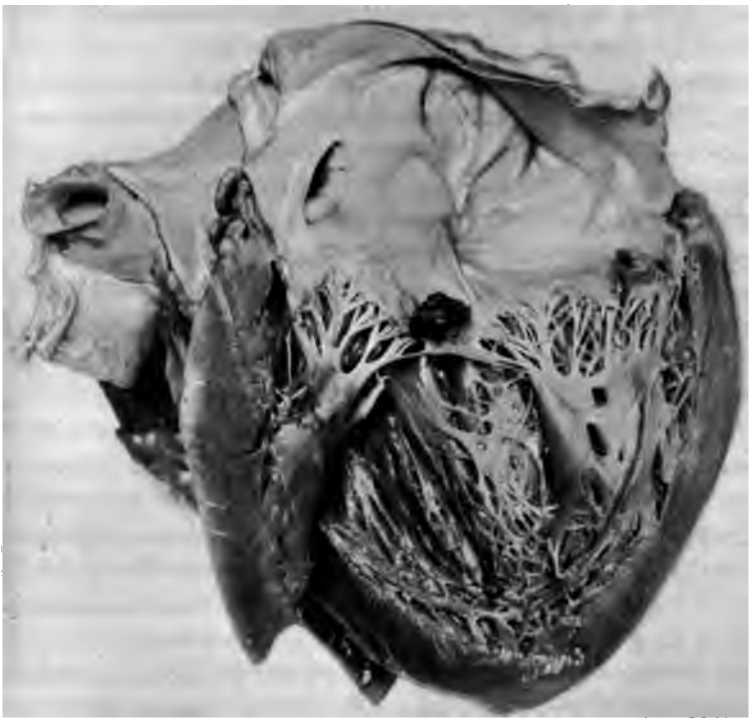


Fig. 25.—Verrucose Endocarditis of Mitral Valve.
Specimen in collection of Dr. Gustav Fütterer.

of *vegetation*. The name is, however, not very appropriate, as the so-called vegetation is in its formation and composition a thrombus, and may contain all the elements of a thrombus, fibrin, red and white blood-corpuscles, and blood-platelets.

By the time that the vegetation has reached such a size as to be noticeable to the unaided eye, the process of repair has begun at its base. This is accomplished by the ingrowth of young connective-tissue cells and the formation of a granulation tissue which finally replaces the entire mass of adherent fibrin, and in time becomes covered by the endothelium from the neighbouring

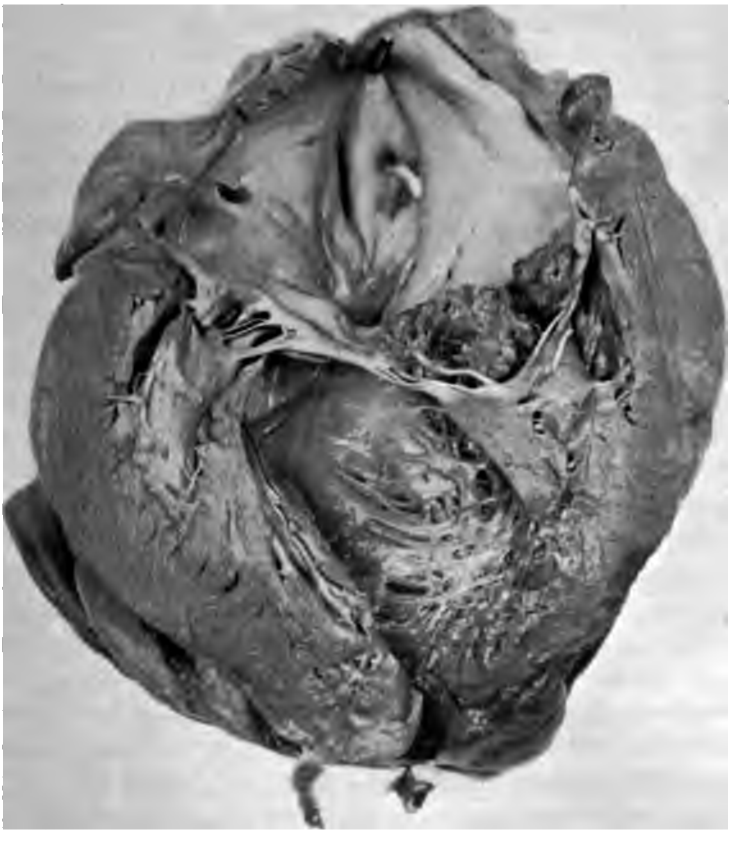


FIG. 26.—MALIGNANT VERRUCOSE ENDOCARDITIS OF MITRAL VALVE.
Specimen in collection of Dr. Gustav Fütterer.

membrane. The growth can now be more properly termed a vegetation, as it is essentially an outgrowth from the subjacent tissue, and some authors limit the term to this form. The accumulation of fibrin over such an affected area may be very large, but the average vegetation is about 3 millimetres in length. When of the irregular form described, the endocarditis is spoken of as the warty or verrucose variety (Figs. 24–27).

The vegetation may be large and polypoid in shape or long and string-like, attached at one end so as to swing in the blood-stream. The disease is then spoken of as of the polypoid or villous variety respectively. The vegetation may be too large for complete organization, and may soften and redissolve in the blood-stream, or portions may break off and be carried in the blood until they reach a vessel of too small calibre to permit their passage, when they plug the vessel and cut off the circulation of the parts supplied by it. The infarcts thus produced by the emboli of simple endocarditis are usually of a non-infective nature.

The further repair of these lesions and the changes in the valves consequent to them are dealt with under the head of Chronic Endocarditis.

The *malignant* form of the disease is mainly characterized by the intensity of the infection, and the fact that embolic phenomena are more common than in the simple, and are almost always of a septic nature. The local lesions may be *vegetative*, *suppurative*, or *ulcerative*, depending on the nature and violence of the infection. In all cases the necrosis of the affected areas is more marked than in the simple form, and ultimately leads to loss of substance, the portions sloughed off passing into the circulation as *septic emboli*.

The valve-cusp thus ulcerated is naturally weakened, and frequently gives way before the pressure of the blood, forming small pouches in the valve, the so-called valvular aneurysms, or giving way completely perforate the valves. Acute valvular insufficiency can thus be produced (Fig. 27). A valve-leaflet may become partially detached, and the free end swing in the blood-stream. Ulceration of the papillary muscles or the chordæ tendinæ may produce stretching or rupture of the cords, or a thrombus covering the affected area may mat them closely together.

When the lesions are situated on the mural endocardium, perforation is possible, and the interventricular septum has been found perforated, or communication has been established between auricle and ventricle, or between the right auricle and the aorta. When the disease is produced by bacteria of suppuration, abscess of the myocardium may result, and, discharging, empty its contents into the circulation. (Septic Emboli.)

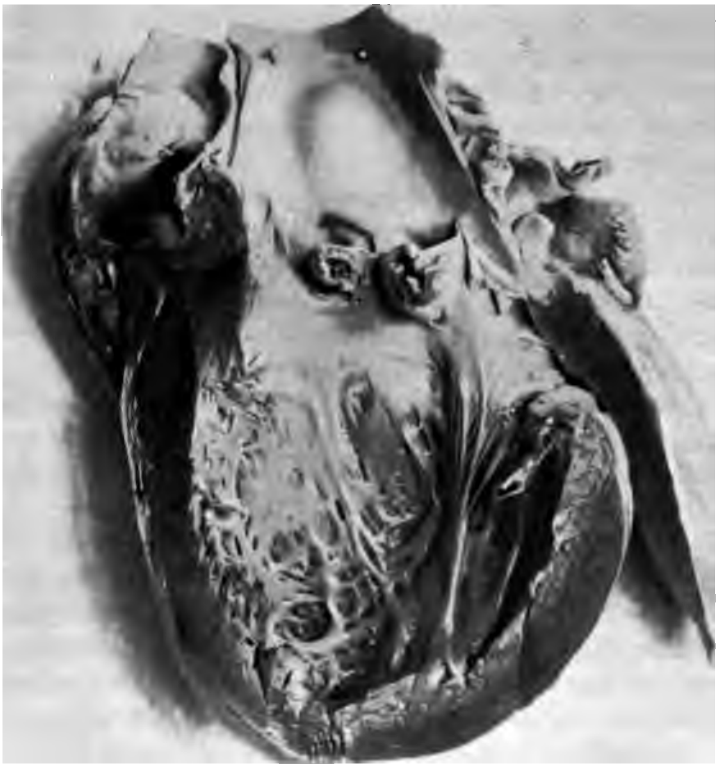


FIG. 27.—MALIGNANT VERRUCOSE ENDOCARDITIS OF AORTIC VALVE, WITH PERFORATION OF A CUSP.

Specimen in collection of Dr. Gustav Fütterer.

Associated with acute endocarditis are found the anatomical changes produced by the disease to which the endocarditis is secondary, since it is extremely rare to find it as an independent disease. It is very often associated with the chronic form of endocarditis.

The secondary changes in the *simple form* are trifling, and as a rule produce no symptoms. It is only in the course of time, when the disease passes into the chronic form, that serious damage is done. Secondary to the *malignant form*, on the contrary, are circulatory disturbances consequent on the ulceration or perforation of the valves, and more important still, the metastatic foci of disease set up all over the body by means of septic emboli. The spleen and kidney are especially apt to suffer in this way. The infarcts so produced may be few, or innumerable *minute foci* of suppuration may be scattered over the whole body. It is to these septic emboli that this form of the disease owes its malignant character.

Etiology.—It may be stated as a general proposition, that the bacterial origin of acute endocarditis, both simple and ulcerative, has been established. Heiberg's discovery in 1872 of micrococci in the thrombotic masses of the malignant form has led to an unbroken series of researches and experiments by the most brilliant pathologists in Europe and this country, with the result that the cloud of doubt and speculation once enveloping this subject has at length been cleared away. Special activity in this work was displayed during the years immediately following 1885, and prominently figuring in this line of investigation are the names of Virchow, Klebs, Birch-Hirschfeld, Koester, Weichselbaum, Fraenkel and Saenger, Rosenbach and Netter in Germany; Gilbert and Lion, Cornil and Babes, Roux, Josseraut, and Dessy, in France; Dreschfeld, Cayley, Purcer, in England; Osler, Flexner, and Prudden in this country. It is manifestly impossible within the limits of this work to give a detailed account of the nature of the researches made by these eminent workers, and it must suffice to state the facts that have been established.

Micro-organisms have been quite generally found in cases of *malignant endocarditis*, some of them being the same as those found in other infectious diseases, a few being specific to endocarditis. Occasionally two or more varieties have existed in the same case. The bacteria most usually discovered have been streptococcus pyogenes, particularly of erysipelas; staphylococcus pyogenes, aureus, and albus, and the micrococcus lanceolatus.

The gonococcus, the bacillus of typhoid fever, of diphtheria, of influenza, and of tuberculosis have also been found, although much less frequently.

Weichselbaum identified certain bacteria, which, because they appear to occur only in endocarditis, he named bacillus endocarditidis griseus and capsulatus and micrococcus endocarditidis rugatus. The bacillus immobilis et fœtidus was also found by Fraenkel and Saenger. That these various bacteria are capable of inducing endocarditis has been shown by experimentation on animals. A number of investigators injected pure cultures of microorganisms obtained from infected valves into the jugular veins of dogs and rabbits, and afterward found these cocci, often in masses, both on the surface and in the deeper layers of both aortic and mitral valves, the valves themselves showing characteristic inflammatory changes. By some experimenters it was asserted that endocarditis could be only thus produced after the valves had suffered trauma by chemical or mechanical irritation. Others, on the contrary, claimed to have produced endocarditis by injection of microbes into animals without previous injury of the endocardium.

In numerous instances the bacteria found on the affected valves have also been identified in the septic emboli thrown off during the course of the disease, while in a few instances the blood of patients suffering from infective endocarditis has been found to contain septic organisms.

The bacteria found in the lesions of ulcerative endocarditis occurring as a complication of typhoid fever and diphtheria are usually pyogenic. This is also true of most cases of gonorrhœal endocarditis, although the gonococcus has been definitely identified in the endocarditic lesions. The pneumococcus of Fraenkel has been frequently found in endocarditis, both simple and ulcerative, but, according to Osler, more frequently in the latter variety.

It appears well established that a primary endocarditis of bacterial origin is occasionally, although rarely, met with. Most instances of endocarditis are secondary to some general or local infection.

There has been considerable speculation, and for a time there was a heated discussion, particularly between Klebs and Koester, over the route by which microbes are carried to the infected valves.

Klebs and Virchow maintained they were deposited on the surface of the cusps out of the blood, while Koester declared they were carried thither in the minute capillaries situated in the deeper layers of the valves. He maintained that the masses of cocci caused embolic plugging of the vessels, which was followed by rupture, thus setting free the bacteria and allowing them to reach the surface. Against this explanation was urged the scarcity of blood-vessels in the valves, as well as the fact that the earliest evidence of inflammatory change is along the line of contact of the cusps. It is now held that both contentions are correct, but Virchow's view is accepted by the majority of observers. The adherents of Virchow's opinion believe that the pressure of the blood forces the micro-organisms between the endothelial cells of the endocardium —a theory that probably accounts for the development of endocarditis in the left heart after birth and in the right side during fœtal existence. As is well known, blood-pressure is greater in the right ventricle before and in the left ventricle after birth.

Another explanation for the localization of endocarditis is that inasmuch as oxygen is necessary to the growth and activity of most bacteria, these organisms are most active in blood relatively rich in oxygen, a condition obtaining in the right cardiac chambers in the fœtus and in the left during extra-uterine existence.

A most interesting question relates to those conditions that determine whether the endocarditis is to be simple or ulcerative, since both forms are of microbic origin, and some of the same organisms have been found in the endocarditic vegetations of both varieties, What are the factors that determine the malignancy or benignity of the affection? It has been suggested that this depends upon the number of bacteria present. It is more probable, however, that when healthy valves are attacked the nature of the endocarditis depends upon the virulence of the infecting organisms. Other factors are of influence, however, aside from the nature or virulence of the bacteria, and these will now be considered.

Simple Endocarditis.—Articular rheumatism is the disease *par excellence* in which acute simple endocarditis is most frequently observed.

Why this is, is not as yet satisfactorily established, but there appears to be a growing belief among pathologists in the bacterial origin of rheumatism, as opposed to the once prevalent notion of its dependence upon lactic acid in the blood. The relative frequency with which these two affections are associated is variously estimated. Of 32 cases of inflammatory rheumatism that terminated fatally, Fagg found the valves affected in all but 12. According to Hayden, Peacock found endocarditis in 16 per cent of his cases of rheumatism, while Fagg puts the ratio as high as 40 or 50 per cent. French as well as English observers put the proportion much higher than do the Germans; thus Bouillaud, 55 per cent; Budd, 48 per cent; Fuller, 23 per cent; while Wunderlich and Lebert give it as 23 per cent, and Bamberger 20 per cent. These differences probably depend upon the severity of the rheumatic attack, since all observers agree in the statement that the valves are far more likely to become inflamed in acute than in subacute or chronic forms of articular rheumatism. Endocarditis is especially liable to occur in a first attack of arthritis, particularly when this is severe and several joints are involved. There is no doubt, however, of its development as a result of subacute or chronic rheumatic manifestations. Hayden is of the opinion that in rare instances endocarditis may be the only manifestation of the rheumatic poison. The period in the course of acute rheumatism at which endocarditis may occur is given by Hayden from the sixth to the ninth day, and by Fuller as from the sixth to the twentieth day. The earlier the time of life at which acute rheumatism develops, the greater is its liability to set up acute endocarditis.

The next most frequent predisposing cause of this form of endocardial inflammation is generally stated to be chorea. Endocarditis of this origin is numerically less frequent than the rheumatic, whereas the relative frequency of chorea and endocarditis is thought by some observers to be greater; thus, of 16 cases of fatal chorea occurring at Guy's Hospital during twenty years, Fagg found post-mortem evidence of endocarditis in 14. Here, again, there has been much speculation concerning the reason of the association between chorea and endocarditis. By some the latter is attributed to acute articular rheumatism, which is now recognised to be frequently associated with chorea. Thus in 40 cases of the latter affection manifesting organic heart-disease, Gowers found in all a trustworthy history of associated rheumatism. There are some observers, on the other hand, who hold that chorea is capable of causing endocarditis independently of associated or antecedent arthritis. Since girls are undeniably more subject to chorea than are boys, endocarditis of this origin is observed more frequently in the former sex.

Scarlatina and measles are also accredited with the causation of endocardial inflammation. This may be either a secondary result, or the endocarditis may be due to a mixed infection. Inasmuch, however, as scarlet fever is not infrequently followed by rheumatic manifestations, the endocarditis is held by some to be referable to the latter and not to the former affection.

Other irruptive diseases, particularly enteric fever and smallpox, are also capable of setting up endocarditis, but as subsequently stated, this is more likely to be malignant than merely simple.

Although gonorrhœa is more likely to cause the ulcerative form, vegetative endocarditis undoubtedly occurs as a result of gonococcus or perhaps a mixed infection, a conclusion that would seem justified by the entire absence of any other etiological factor in certain cases of valvular disease.

As previously stated, there is both clinical and pathological evidence of the occurrence of acute endocarditis in the course of croupous pneumonia or in consequence of pneumococcus infection. Although such an endocarditis is more likely to be ulcerative, it may nevertheless be benign. In a fatal case of pneumonia, which had exhibited no evidences of endocarditis during life, Haushalter found a colony of pneumococci in the interior of one of the mitral cusps, while the other showed an almost invisible swelling of the endothelium near the point of insertion of the valve. From this he concluded that not only is acute endocarditis a probable sequence of pneumonia, but also endocarditic changes of a slow sclerotic type may be ultimately set up. His conclusions were as follows: (1) The absence of murmurs during life or of naked eye changes post mortem does not prove the integrity of the valve, since during the course of the pneumonia the pathogenic organisms may be carried into the interior of the valve, and thus prove the starting-point of future valvular mischief. (2) The possibility of such an endocarditis should be remembered, since a latent period may exist between the primary disease and the development of the endocarditis. (3) The possibility of such an occurrence renders it advisable for the physician to keep a patient under prolonged observation after recovery from pneumonia, and to make repeated examinations of the heart, that he may thereby detect the earliest manifestations of a valvular lesion.

Injury of the valves through strain is generally recognised as one of the conditions predisposing to the occurrence of acute endocarditis. By some it is contended, however, that strain alone is not sufficient, but must be united with some previously existing defect. Strain is probably capable of setting up fresh inflammation of a valve, already the subject of a former though slight endocarditis. Rupture of a cusp may undoubtedly prove a starting-point of acute inflammatory change.

Age is an undoubted predisposing factor, acting in most cases, however, in the way of rendering individuals susceptible to articular rheumatism, the exanthemata or other diseases, themselves capable of bringing about endocarditis. Supporting this view or interpretation of the influence of age is the statement that rheumatism is more likely to occasion endocarditis in childhood than in the later periods of life. Both sexes are liable to endocarditis, yet according to some this affection is more frequent among females, although males are said to be more subject to articular rheumatism. To my mind there is nothing in sex, *per se*, rendering the endocardium more vulnerable in females than it is in males.

Females are more subject to chorea and, by reason of their sex, to puerperal septicæmia and infections from pelvic disease, and hence it may well be that they furnish a greater numerical proportion of cases of acute endocarditis, benign as well as ulcerative. With increasing experience, I find myself growing in the conviction that hereditary influence plays a not unimportant rôle in the development of endocardial disease. Cardiac, and particularly valvular lesions, as such, cannot be inherited, but it seems to me that in some families whose members evince pronounced rheumatic diathesis, there is an inherent vulnerability, possibly hereditary, of the endocardium in the presence of rheumatism.

Ulcerative Endocarditis.—It is a well-known fact, established both by clinical and post-mortem observation, that the malignant form is particularly prone to develop as the result of fresh bacterial invasion in valves already the seat of chronic endocarditis or sclerotic change. This is particularly true of the aortic-valve apparatus.

Infectious endocarditis is also specially liable to attack individuals suffering from exhausting diseases or cachexiæ, chronic alcoholism, cirrhosis of the liver, hepatic abscess, cancer, etc.

General infection, as pyæmia, puerperal septicæmia, influenza, diphtheria, variola, and localized septic processes or abscesses, predispose to malignant rather than to simple endocarditis. Flexner found staphylococcus aureus in a case of endocarditis in which the atrium of infection was leg ulcer; in another, staphylococcus, the point of entrance being the intestine; in still another, streptococcus and staphylococcus, the atrium being hepatic abscess. This form of acute endocarditis has been particularly frequent in association with puerperal septicæmia due to infection either of the uterus or its adnexa. It has been known to follow tonsillitis, and even so apparently trivial a local process as a furuncle.

Croupous pneumonia or a pneumococcus meningitis has not infrequently been found as the primary infection in cases of ulcerative endocarditis. Although the pneumococcus may occasionally give rise to the simple form, it is much more frequently responsible for ulcerative inflammation of the valves. This malignant form is also stated by Dreschfeld to have been associated with 7 cases of gall-stones "with or without suppuration of the biliary passages." Dreschfeld thinks that the discovery of the bacterium coli commune in diseases of the biliary passages may explain their connection with acute endocarditis. In this connection it is interesting to note that Flexner, in one case of endocarditis associated with carcinoma of the pylorus, identified the bacillus coli, together with the bacillus pyocyaneus; and Hasenfeld has reported artificially produced endocarditis in animals infected with the bacillus pyocyaneus. Of further interest is the fact that, despite the severe infectious process, distinct hypertrophy of the heart was observed to develop in so short a time as a week.

Finally, this form of endocarditis, although much less frequently than the simple, may exist in connection with articular rheumatism, as mentioned by Osler and others.

Its dependence upon the diphtheria bacillus, though rare, is undoubted. Howard has reported a case in which a bacillus was discovered identical in all respects with the Klebs-Loeffler bacillus. It is now generaly recognised that septic endocarditis, although capable of being set up by pathogenic organisms, is most frequently caused by streptococci and staphylococci.

Symptoms.—Just as it is sometimes difficult from a pathological standpoint to say whether the soft, easily detached thrombi belong to the vegetative or infective variety, so a sharp dividing line cannot always be drawn clinically between the two forms of endocarditis. Nevertheless, I think it will conduce to clearness if, as is usually done, they are described separately.

Acute Simple Endocarditis.—As this process frequently occurs in the course of articular rheumatism, of which it may be regarded as a *manifestation, and not a complication*, its symptoms are often masked by those of the arthritis. If the endocarditis be of a mild type, it may pursue a latent course, and only be detected by its results when years subsequently the individual is found to have a valvular lesion, probably dating back to some almost forgotten rheumatic attack. Such a possibility should always be borne in mind by any physician attending a case of articular rheumatism, however mild, and should incite him to a daily examination of the heart, since many times acute endocarditis is only recognisable by such means.

If in the course of rheumatic fever, especially towards the end of the first week, the temperature unexpectedly rises, and cannot be accounted for, by involvement of a fresh joint or some complication, attention should be at once fastened upon the heart. If the endocardium has become inflamed, with or without implication of the pericardium, the fact will eventually declare itself by the physical signs subsequently to be described, even though subjective symptoms are wanting.

In some cases subjective symptoms become manifest from the start, and are then due probably either to the severity of the endocarditis or to associated myocarditis or pericarditis. These symptoms are præcordial pain more or less pronounced, or an ill-defined sense of oppression and discomfort in the cardiac region, palpitation, the heart-action in some cases being quite tumultuous, and particularly a *subjective sense of dyspnœa*. By this term is meant a sensation on the part of the patient of air-hunger, which may not be evinced by laboured or hurried respiration, but which is usually greater than can be accounted for upon examination of the chest. I cannot now recall a single case of acute endocarditis, recognised as such, in which this symptom was not present. In some instances this feeling of breathlessness actually amounts to orthopnœa; in others dyspnœa is paroxysmal, compelling the patient to sit up in bed during the continuance of the paroxysm. In mild cases the pyrexia is likely to be mild, and possesses no peculiar character.

In other instances the disease produces profound constitutional disturbances, with fluctuating fever and profuse perspiration, plainly suggesting infection, and with a pulse so empty, irregular, and perhaps accelerated, as to at once direct the physician's attention to the heart. These are the cases difficult, if not impossible, of differentiation from the malignant form.

Embolic phenomena are less frequent in the benign than in the malignant form, yet when embolisms occur the symptoms they induce are referable to mechanical interference with the circulation, rather than to a local or general septic process. The most usual seat of infarcts is in the kidney, intestines, and brain. They undoubtedly occur many times without giving rise to recognisable symptoms; yet when such are produced, they are a sudden, sharp pain in the affected part or organ, chill more or less pronounced, and pyrexia. If the embolus lodge in a kidney the urine is likely to contain blood, albumin, and even pus. Hemiplegia and aphasia, the result of cerebral embolism, may in rare instances furnish the first, or perhaps the conclusive, evidence of the existence of acute endocarditis. The following case is instructive: W. J. M., male, aged forty-eight years, height six feet, weight 176 pounds, first consulted me November 9, 1896, not, he said, because he thought himself in poor health, but because, having some heart-trouble, a friend advised him to get my opinion. Family history was unimportant in its bearings upon the patient's condition, but it was stated that one sister had died of consumption, another of insanity, and a third, then living, had heart-disease. Patient declared that he had not been ill since his twelfth year, but had had syphilis at the age of twenty. After his death, it was stated by his wife that the patient had known for seven years of the existence of some sort of heart-disease. Symptoms such as dyspnœa and palpitations were denied, but the patient, when questioned regarding pain, said, " Once in a while a little, down near the heart."

The pulse was noted as 89, not distinctly collapsing, the left seeming slightly smaller. Carotids and subclavians throbbed strongly. Apex-beat in sixth left intercostal space, 3¾ inches from the sternum, and the cardiac impulse was heaving; diffused from the fifth to the seventh, but maximum in the sixth interspace. Absolute cardiac dulness was practically normal, but the relative was increased to the left, extending 5½ inches to the left of the breastbone, downward to the seventh rib, and but 1 inch to the right of the sternum (Fig. 28). The first sound at apex was muffled and the second was wanting; throughout the præcordium the sounds were obscured by murmurs, both systolic and diastolic, which were audible over the entire cardiac area, but were of maximum intensity in the aortic area and on the body of the sternum. A snapping systolic tone was audible in the femoral artery. In the aortic area bimanual palpation with slight pressure brought out a systolic shock and thrill. Examination of abdomen and urine was negative.

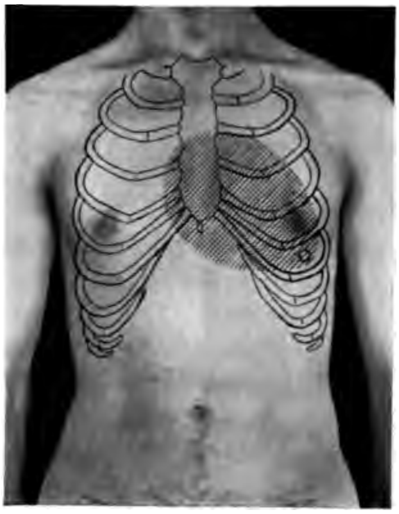


FIG. 28.—APEX-BEAT AND RELATIVE DULNESS, CASE OF ACUTE ENDOCARDITIS (p. 158).

The diagnosis lay between aneurysm of the ascending aorta and insufficiency of the aortic valves, but the lesion was subsequently decided to be a valvular one of sclerotic, possibly syphilitic origin.

For the next few months the patient was seen at rather infrequent intervals until the last of March, 1897. In February of that same year patient was knocked down by a runaway horse, but did not think he sustained special injury. Towards end of March he began to complain of insomnia, great nervousness, and restlessness. The heart was rapid and pounding, and there was dyspnœa, even in repose, which increased paroxysmally without cause. Urine analysis showed pus, blood, and albumin.

Patient was ordered to keep to the house and confine himself to milk diet, with potassium citrate and tincture of digitalis in small doses, with saline cathartics daily. After about two months the urine lost all traces of blood and albumin, but patient's general condition grew worse, and he was ordered to keep his bed. Heart's action was still rapid and pounding, but regular, and dyspnœa with paroxysmal exacerbations very marked. Patient sweated profusely, but the thermometer never showed fever.

One night complained of pain in right hypochondrium below ribs, embolism was suspected, but subsequently doubted. Liver reached 3 fingers below costal arch, was moderately tender, firm, and with rounded border. The condition was thought to be passive congestion without infarction. About the last of May patient developed mental symptoms, as shown by ugliness of temper, especially towards wife; it appeared to be a mild acute mania, and the wife stated that the mother as well as a sister had died insane. Hyoscine hydrobromate was ordered, supplemented subsequently by valerianate of ammonia, with improvement, the delirium being only occasional and ugliness less.

June 2, 1897, examination showed the following: Radial pulse distinctly collapsing, venous pulsation in forearm, but external jugulars not turgid and without pulsation. Apex-beat in sixth left interspace, anterior axillary line, systolic impulse in second and third right interspaces near sternum, followed by diastolic thrill, also a more feeble pulsation in fourth, fifth, and sixth right interspaces, slight systolic shock in second left interspace near sternum. Absolute dulness, patient in dorsal decubitus, reached 6 centimetres to right of median line, and from second to sixth costal cartilage, the note being flat, with marked resistance from second rib to fourth interspace, and slightly less dull below this point. Dulness also reached 10.5 centimetres to left of median line (Fig. 29).

Auscultation showed first sound at apex, dull and muffled, but no distinct murmur, a double tone audible below left clavicle and down along left axillary line; a systolic tone and soft diastolic murmur in second left interspace and outward 1½ inch from sternum. There was also a faint second sound in the pulmonary area, and when the patient took a deep inspiration and held his breath the second sound seemed to be changed into a soft murmur.

The soft diastolic murmur at left of sternum was transmitted faintly downward. A painfully loud and harsh diastolic and systolic murmur was heard in second and third right interspaces, and transmitted more feebly into fifth, out to nipple and up to neck.

The condition was interpreted as follows: Acute endocarditis ingrafted on a chronic endocarditis, affecting aortic valves and aorta, and producing dilatation of this vessel. No evidence could be found of inflammation of other valves, and yet the great extent of dulness to right of sternum was thought due, in addition to aortic dilatation, to dilatation of the right auricle, secondary to mitral insufficiency. Cough was at no time a marked symptom, except two or three paroxysms a few hours before death, when patient seemed to have pain in left lung. During the last few weeks of life there was moderate œdema of ankles and shins, also puffiness, but no pain, in left wrist and hand. Forty-eight to sixty hours before death patient became comatose, with cold extremities, very rapid, feeble, and irregular pulse, and the trunk and lower extremities became studded with small brownish-red spots, that had all the characters of cutaneous embolisms. Death, which took place June 24th, seemed to be the result of gradual cardiac asthenia.

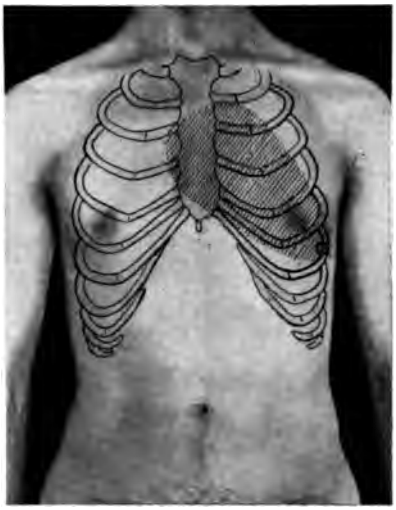


Fig. 29.—Apex-beat and Absolute Dulness Later in Same Case as Fig. 28.

The autopsy, made by Dr. W. A. Evans thirteen hours after death, was briefly as follows: Very large numbers of petechiæ over abdomen, chest, and legs, about the size of a pea. Some interstitial splenitis and perisplenitis and zones of connective-tissue growths representing old infarcts. These were generally subcapsular.

Liver.—Fatty infiltration—nutmeg. Large numbers of small islands of connective-tissue increase, quite generally distributed in subcapsular zone. A small mass of calcareous material in lower portion of right lobe, superficial. In left lobe a small fresh infarct about 6 millimetres in diameter.

Left Kidney.—Slight parenchymatous nephritis.

Right Kidney.—In the cortex an old infarct 1 centimetre in diameter, over this the surface of the kidney depressed. This infarct, fatty in appearance, reddish, surrounded by a reddish zone. It was this infarct which in March had occasioned the bloody and albuminous urine.

Left Pleural Cavity.—No fluid, no adhesions except to diaphragm.

Left Lung.—Congested and œdematous. In anterior edge of inferior lobe an apoplectic focus about 1 centimetre in diameter, quite recent.

Right Pleural Cavity.—Extensive old, firm, fibrous adhesions quite general.

Right Lung.—Congested and œdematous, single hæmorrhagic infarct 2 centimetres in diameter.

Pericardium.—No effusion, uniform adhesions between pericardial layers. They strip easily, appear gelatinous or mucoid.

Aorta.—Tubular dilatation of aorta in its first portion. The lumen is somewhat ovoid, measuring 9 by 8 centimetres. The aortic ostium dilated with compensatory stretching of the aortic cusps. The aortic cusps, measured along their free edge, show a length of 5 centimetres, 4½ centimetres, and 3½ centimetres respectively. All of the cusps show ridges of atheroma, with considerable thickening and stiffening. At the base of the largest cusp, a calcareous plate. Thickening, redness, and some deposit of fibrin on each of the cusps, especially towards the free edge.

The valves not competent. Aorta atheromatous. Areas of calcification, atheromatous ulcers, and some vegetations around these losses of substance. The left ventricle enormously dilated, its wall 3 centimetres in thickness at its thickest portion. Myocardium is not especially fatty. Mitral valves show multiple foci of acute endocarditis, consisting of small, round red masses, the size of a pin-head.

Left auricle very much dilated, right heart otherwise normal.

Cultures made from the vegetations give no growth of microorganisms.

Diagnosis.—Tubular aneurysm first portion of aorta; atheroma of aorta and aortic cusps; hypertrophy and dilatation of the left heart; acute endocarditis and endaortitis, vegetative in character. Recent infarcts in liver and lungs.

This case illustrates the proneness of acute inflammation to attack valves that have undergone sclerotic changes. It is probable that the aortic regurgitation diagnosed in the fall of 1896 was due partly to incompetence of the cusps from stiffening and rigidity and partly to stretching of the ring consecutive to the dilatation of the ascending aorta, the valves not being able to adequately close the ostium in spite of their compensatory stretching. With the exception of the lack of febrile temperature the symptoms strongly suggested ulcerative endocarditis, and show how difficult and unwise it is to attempt a sharp clinical distinction between the two forms of endocarditis. The anatomical changes were those of the vegetative variety, and yet in its rapid course and fatal termination the process may be said to have been malignant.

Course and Termination.—As already stated, in some cases rheumatic endocarditis of a mild type may be easily distinguished from infective endocarditis, while other cases seem to occupy intermediate ground, and clinically, at least, cannot be classed with either one or the other type. It is plain, therefore, that the course and termination are equally variable.

Simple rheumatic endocarditis may pursue a favourable course, and terminate in the recovery of the patient, nay, may even subside without serious impairment of the affected valve. In the majority of cases, however, the patient is usually left with a chronic valvular lesion.

Ulcerative Endocarditis.—Under this head are reckoned those cases of inflammation of the endocardium which manifest more or less pronounced symptoms of general sepsis, whether the termination is in death, by far the more frequent occurrence, or in recovery, of which instances are now and then reported. As might be expected from a consideration of the etiology and morbid anatomy of this class of cases, the clinical picture varies much, according as the local—that is, cardiac—or the general symptoms predominate. In the majority of cases the symptoms are those of general sepsis, with very subordinate, or it may be with no manifestations on the part of the heart. In such cases the inflammatory changes in the endocardium are to be regarded as *merely an incident* of the general infection, and therefore Rosenbach classifies these cases as merely local manifestations of a general infection. The endocarditis is but one of the many possible local expressions of the infection, in consequence of the profound disturbance of nutrition there induced.

In this class of cases the conspicuous features are phenomena characteristic of pyæmia, an irregularly continuous pyrexia, with few if any rigors, sweatings, great prostration, anæmia, emaciation, anorexia, diarrhœa, a dry, brownish tongue, abdominal distention, stupor or low muttering delirium, persistent dorsal decubitus, and enlargement of the spleen. The *pulse* is only moderately accelerated, in most instances impressing one as being chiefly remarkable for its feebleness and want of tension, while the heart may display absolutely no evidence of disease, or may be slightly dilated, with a faint, soft systolic apex or basic murmur, the same as in typhoid fever. Indeed, this whole condition is so like enteric fever as to be frequently, it may be said usually, mistaken for that disease.

In other cases the fever is much less typically septic, remitting or intermitting, not dropping suddenly below normal, and again abruptly shooting up several degrees, but running so mild a course as to scarcely merit the appellation of pyrexia.

In others, again, the elevation of temperature is of irregular type, or there are diurnal fluctuations, to possibly 101.5° or even 102.5° F. The feature that mainly attracts attention in such cases is the progressive anæmia, and the trifling changes discovered in the heart are commensurate with those of anæmia.

The last of July, 1900, I was consulted by a German, aged fifty-eight, who was a merchant in the interior of Indiana. His family history was unimportant, and his personal anamnesis was meagre. He had always considered himself well until about a year previously, when he had suffered from bronchitis, for which he had received medical treatment, at which time a heart-murmur was discovered. In March, 1900, he had been troubled with night-sweats that had resisted treatment by belladonna. Since that time he had been losing ground, and altogether his weight had declined from 180 to 137 pounds. He gave a vague account of some gastro-intestinal disorder in the spring, but could not recall any attack of pain that might have been an attack of appendicitis, hepatic or renal colic. Neither could he remember any injury or local suppurating process, and he had never had rheumatism, pneumonia, gonorrhœa, or other infection.

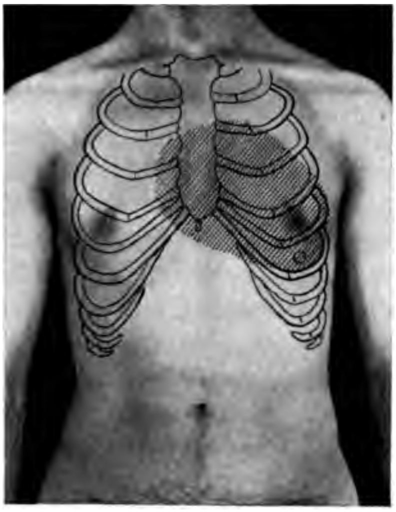


Fig. 30.—Apex-beat and Relative Cardiac Dulness (p. 164).

The patient was tall, emaciated, pale, and of a slight yellow hue, and his conjunctivæ were faintly icteric. The radial arteries were thickened, and the pulse, of only moderate tension, was regular and equal, 100 to the minute. A feeble cardiac impulse was diffused from epigastrium to the apex-beat, which, weak and accompanied by soft thrill, was situated in the sixth interspace, 10 centimetres to left of median line. Relative cardiac dulness at the level of the fifth costal cartilage extended from 5 centimetres to right of the midsternal line to 14 centimetres to left of the same (Fig. 30). The first sound at the apex was obscured by a murmur and the second was impure, but over right ventricle both were more distinct, while at the base both were faint, the second being scarcely audible, the pulmonic second the louder of the two; both aortic

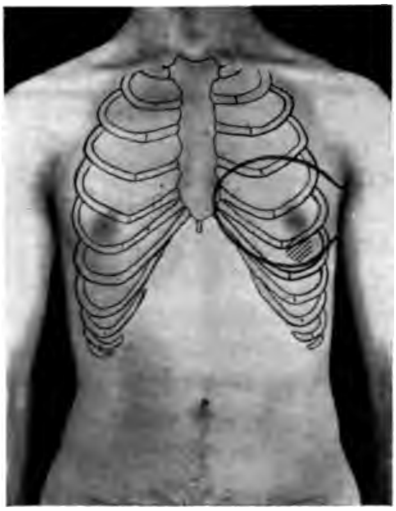


Fig. 31.—Area of Maximum Audibility (shaded) and Transmission of Murmur in Case (p. 166).

tones were distinct, but feeble in the cervical arteries. A harsh systolic murmur was heard at the apex, and was transmitted into the middle of the axillary region and to the median line in front, yet not above the third interspace (Fig. 31). The lungs were negative and the abdomen was flabby, moderately tympanitic, not tender, while in the location of the gall bladder a soft roundish body could be plainly made out. The urine collected over night and analyzed next day gave following results: Quantity, 800 cubic centimetres; cloudy, specific gravity, 1.015; reaction acid; colour, reddish-yellow; urea, 1.4 per cent; mucin present; a slight trace of albumin; no sugar; no bile; no blood; 2 granular and a few hyaline casts; a few cylindroids and uric-acid crystals, but no pus; not examined for peptone. The blood-examination on that day showed hæmoglobin, 40 per cent; red cells per centimetre, 3,666,230, percentage of red cells, 73.3; corpuscle index, 54.4, and number of leucocytes per centimetre, 13,700. His temperature at 11.15 A. M. was 98.4° F.

As there was nothing in the examination thus far to lead to a suspicion of endocarditis, the diagnosis was made of chronic arteriosclerosis, with chronic myocarditis and an atheromatous mitral incompetence and a mild interstitial nephritis, anæmia. The patient was advised to go into a hospital, where he could receive treatment by rest, appropriate diet, and medication. He did as advised, but got chilled in driving to the hospital, and at my visit that same afternoon his temperature was found to be 101.8° F., respirations normal, and pulse only moderately accelerated. This febrile reaction was thought to be the result of his chill, and fearing he might develop uræmia on account of his nephritis, he was ordered to drink very freely of hot water, so as to promote elimination. The result was that by 6 P. M. he had passed 15 ounces of urine, and his temperature was normal. Believing this rise of temperature to have been but a transient flurry, I was surprised and disappointed to find that later that same evening his temperature again rose to 101.4° F. The chest was then gone over carefully, but with negative results, and accurate record was kept of the amount and character of the urine, also without the discovery of anything of importance.

During the succeeding six days, as shown by the annexed chart (Chart I), he had a low morning temperature and a moderate afternoon pyrexia with slight perspirations, but no rigors, and he asserted that he felt well. As the patient had come from a State in which malaria is common, his blood was examined on August 1st for plasmodia, but with negative results. Nevertheless it was decided to test the effect of quinine, and on August 2d 20 grains were exhibited in the early morning. On that day he had no fever, but on the next day, without quinine, his temperature went up as usual to 101.4° F. On August 4th 15 grains kept it down to 100° F., and on the 5th to 100.8° F., while on the 6th, without the remedy, the temperature again reached 101.8° F., and with 20 grains on the 7th it did not go over 99° F. On the 8th,

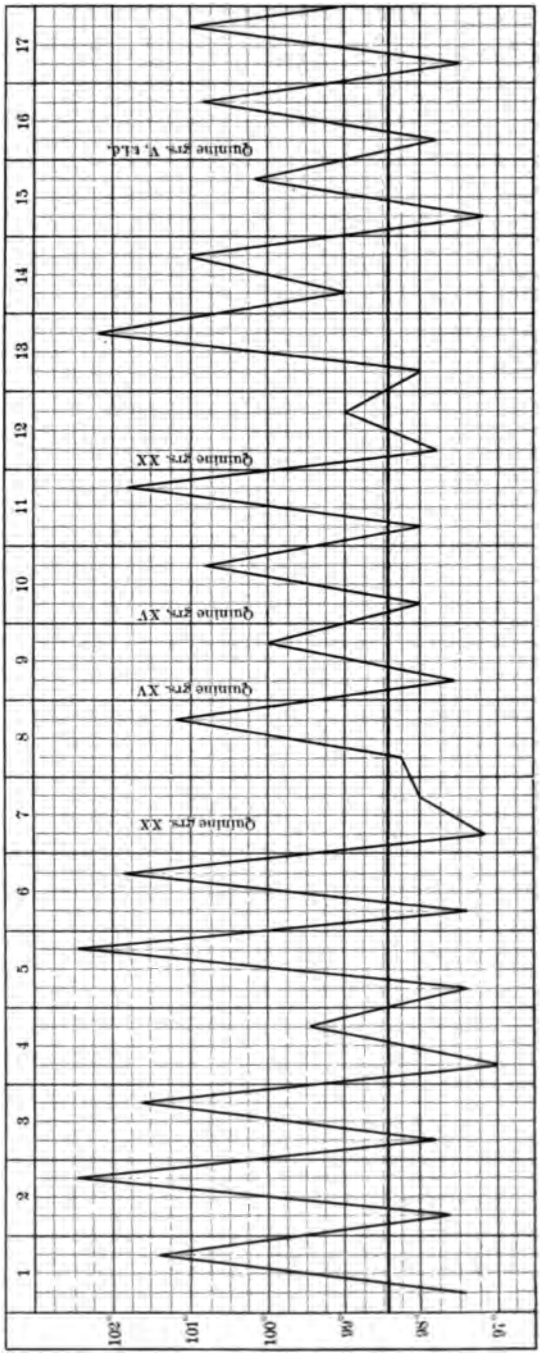


CHART I.—TEMPERATURE IN CASE OF ACUTE ENDOCARDITIS (p. 166).

without quinine, his temperature was 102.2° F., on the 9th a degree lower, while 5 grains t. i. d. on the 10th kept fever down to 100.2° F.

The results thus obtained convinced me that the quinine acted as an antipyretic, but that, in spite of doses sufficient to exert a more lasting influence in case the symptoms were due to malaria, the fever reasserting itself so soon as the drug was stopped, there was some other cause at work. The urine was now tested again for peptone, and with positive results. I therefore became satisfied that the case was one of pyæmia. It should also be stated that the blood had been tested for the Widal reaction, and this proved to be absent.

As the gall bladder had been found enlarged at the first examination, and was still palpable, it was thought highly probable that there might be an empyema of this receptacle as the source of the infection. Accordingly the patient was seen by Dr. Bayard Holmes, who corroborated the gall-bladder enlargement and concurred in the opinion of a probable empyema. This seemed all the more likely to be the focus of infection from the fact that no disease could be discovered in the rectum or elsewhere. A few days subsequently Dr. Holmes operated on the gall bladder, but although it was enormously distended with fluid, this was not purulent, the condition being a catarrh and not an empyema.

No ill effect followed the operation, but the wound healed very slowly. For a day or two the temperature fell somewhat, but then resumed its former characters, being down nearly or quite to normal in the early part of the day and rising in the afternoon or evening to between 101° and 102° F. Rigors were never manifested, and repeated queries concerning chilly sensations always elicited a positive denial of them. Perspirations were also absent, or at the most amounted to no more than a scarcely perceptible moistness of the skin where covered.

Week by week the patient lost in strength and flesh, and grew more anæmic in appearance, although his ability to take considerable nourishment persisted. He also displayed slight apathy, lying flat in bed, and changing position but seldom. Pain was not complained of, and in vain was a daily search kept up for signs of embolism. During these weeks the pulse slowly and gradually became a little more accelerated, reaching 116 or thereabout. Its striking feature was its feebleness. This want of tension, together with the growing weakness of the heart-sounds, induced me to administer strychnine in doses of $\frac{1}{30}$ of a grain 4 times daily, along with 5, and afterward 7 drops of tincture of strophanthus thrice daily. He was also given moderate doses of wine and whisky.

It was always difficult, owing to the rigidity of the chest-wall and the voluminousness of the lungs, to satisfactorily map out deep-seated cardiac dulness, but I became convinced that the heart gradually increased somewhat in size, though no more than could be attributed to myasthenia. *Pari passu* with the increasing feebleness of the heart-sounds the systolic apex-murmur augmented in intensity and extent of audibility until at length it spread throughout the entire præcordium. It remained to the last a rather harsh blowing murmur, and no new bruits ever developed. Neither did œdema become more than a very slight pitting over the tibiæ, and I could not make out any increase in the size of the liver or more than a trifling increase in the area of splenic dulness, the organ not being distinctly palpable.

In brief, the entire clinical history was that of an ever-growing anæmia, or better, marasmus with moderate intermittent pyrexia. In this respect I should think it a fair example of the type of cases described by Romberg as anæmic rather than markedly septic, and yet there was no doubt of the existence of a mild pyæmia. Peptone was repeatedly found in the urine, and thus, with the increased leucocytosis, confirmed the conclusion drawn from the temperature record. This is well shown by the annexed chart. Death occurred the middle of September, about eight weeks after he came under observation. No autopsy was had, but I believe this was a pyæmia, with implication of the endocardium as a secondary and not a primary event.

In still other cases of ulcerative endocarditis there are rigors, sudden high elevations of temperature, and profuse sweating, followed by sharp decline of the pyrexia, the picture not unlike the paroxysms of malaria, excepting that the septic phenomena lack the periodicity of malarial infection. Murchison has narrated an instance of this kind that lasted three months, and was so suggestive of malaria to the friends that in deference to their wishes quinine was freely administered, but without appreciable effect on the disease. In his case, however, there was physical evidence of an aortic valvular lesion.

There are other cases, again, in which the temperature pursues a still more erratic course, rising and falling abruptly for days, and then suddenly sinking to normal, or it may be to below normal. Remaining thus for days or even weeks, it again assumes its former irregularly intermittent type. During the apyrexial period the pulse still remains conspicuously feeble, and the patient fails to regain strength, so that by these symptoms it becomes manifest that actual improvement is not taking place.

Thus, whatever the various manifestations, they are in themselves indicative of sepsis, and there is nothing to show that the heart has become affected. Occasionally, on the other hand, the involvement of the endocardium is evinced by the appearance of cutaneous infarcts or by phenomena of embolic plugging of vessels in the intestinal organs. The lodgment of emboli in the skin is shown by the appearance on the extremities or trunk, still more rarely upon the neck and face, of reddish spots of variable size, from that of a pin's head to a pea, or somewhat larger. These petechiæ may be few and scattered irregularly, or they may come in showers and at irregular intervals. Septic infarcts in the liver, spleen, kidneys, etc., are productive of abscesses, which may be miliary and escape detection during life or be of recognisable size. In some instances the clinical picture is dominated by these embolic phenomena.

I recall a case in which it was the detection of a splenic abscess which seemed to justify the diagnosis of septic endocarditis. I was asked by Dr. Haskin, of Highland Park, to see a gentleman of about sixty who had been having irregular chills, fever, and sweatings suggestive of malaria. His illness had begun six weeks before with malaise, and had speedily developed the symptoms of sepsis. Three days prior to my visit he had suddenly complained of sharp pain in the left hypochondrium with tenderness. I found him unconscious, and lying turned to the left side, with his thighs flexed, skin hot and moist, pulse moderately rapid, but notably weak and soft. There was a distinct blowing systolic murmur in the mitral area. Splenic dulness was greatly increased, and although palpation evidently caused pain and was resisted, a rather soft tumour having the form of the spleen could be felt extending well down below the left costal margin. The case was thought to be an instance of abscess of the spleen, probably secondary to acute ulcerative endocarditis. Death took place a day or two later, and, although an autopsy could not be obtained, permission was granted to make an abdominal incision for the purpose of verifying the existence of the abscess. The abdomen was opened accordingly, and so soon as the doctor's finger pressed upon the spleen the organ burst and pus welled up over his finger. In this case no etiological factor could be obtained, and yet it seemed plainly one of septic endocarditis with infective infarct in the spleen.

When emboli of this character lodge in the kidney, they are declared by albumin, pus, and blood, with casts in the urine. There is a class of cases characterized by Romberg as atypical which run their course with all appearance of an acute hæmorrhagic nephritis, albuminuria, pus, blood in varying amounts, casts, renal epithelium, and cylindroids.

This condition of the urine persists throughout, but with remissions and exacerbations. A mildly irregular, continuous fever accompanies the nephritis, and the patient displays pronounced and increasing anæmia. It is this feature, together with the persistent pyrexia out of proportion to the temperature usually observed in nephritis, which throws light on the nature of the case. The discovery of an old-standing valvular lesion contributes greatly to the establishment of the diagnosis.

Lastly, cases are encountered which display such manifest cardiac symptoms that by some clinicians they are classed in a special group. These are such as either develop upon a chronic valve-lesion or occasion such rapid ulceration and destruction of the valves that objective cardiac phenomena become apparent. Symptoms of general sepsis may not be marked, or if pronounced, depend largely upon infective emboli. The pulse is peculiarly soft, regular or irregular, and more or less accelerated out of proportion to the degree of fever. The patient usually manifests dyspnœa, and it may be cyanosis. Both liver and spleen are enlarged from congestion or infarcts. Exceptionally one may upon repeated examinations of the heart detect some evidence of changes going on, such as increasing feebleness of impulse, slight increase in the area of dulness, and perchance a soft murmur where previously none existed, or an alteration of one already present. More often such examinations are futile, and the diagnosis is largely conjectural or has to be made from the symptom-complex.

Course and Termination.—The duration of acute ulcerative endocarditis is exceedingly variable. The disease may run its course to a fatal termination within a few days from its coming under observation, or its course may be dragged out over a period of weeks and even months. Some cases progress steadily to a fatal issue, while others show periods of seeming improvement, followed by exacerbations and more rapid decline. When death is the result it occurs either in consequence of local changes in the heart and cardiac asthenia, or as a result of acute pulmonary œdema or pulmonary infarcts. In other cases the patient is worn out by prolonged sepsis, or death is directly attributable to the effects of embolism in the brain, kidneys, spleen, etc.

A gentleman of about forty, who had a perfectly compensated mitral regurgitation of rheumatic origin, became ill in the fore part of February with what, from the history, appears to have been an acute follicular tonsillitis. It subsided in a few days, but the man did not regain his accustomed health. He felt weak and slightly feverish, had vague pains, dull headache, and lost appetite. Thinking his liver was at fault, he went to French Lick Springs, Indiana, and there drank the waters, took a course of baths, and exercised, but without improvement. While there he was annoyed by tenderness and pain in the ends of his fingers, and observed that at such times there was a faint reddish colour of the skin immediately above the roots of the nails. He was told it was rheumatic.

At length, failing to regain health, he returned home, and towards the end of April sought my opinion, partly on account of a frequent dry cough that had recently developed. I had known him in health, and had previously examined his heart. The change in his appearance and general condition shocked me. His voice was weak and tremulous, his hands shook, his face was pale and sallow. His eyes were not perfectly clear, the skin of his arms and trunk was flabby, faintly yellow, and although at first dry, became bedewed with moisture upon the exertion of undressing. His temperature in the mouth was 99.8° F., and the pulse of 105 was weak and small, but regular. I was struck by the fact that its rate did not fall appreciably, even while he was resting on the lounge after my examination. The heart did not seem to be enlarged, but the apex-beat was feeble and preceded by a short, scarcely perceptible thrill.

The old mitral systolic murmur was present, but in addition the first sound had a suggestion of a presystolic bruit, and the second sound was feeble. The lungs were negative, the liver barely palpable, and urine contained a trace of albumin with granular and hyaline casts.

From the history of tonsillitis and the symptoms, I at once suspected endocarditis, and sent him home to bed. His temperature, which was carefully recorded four times daily, showed a fairly continuous pyrexia, from about 100° F. to 101° F. and a fraction (Chart II). His pulse remained persistently in the neighbourhood of 105, and the heart-findings were unchanged. He was given

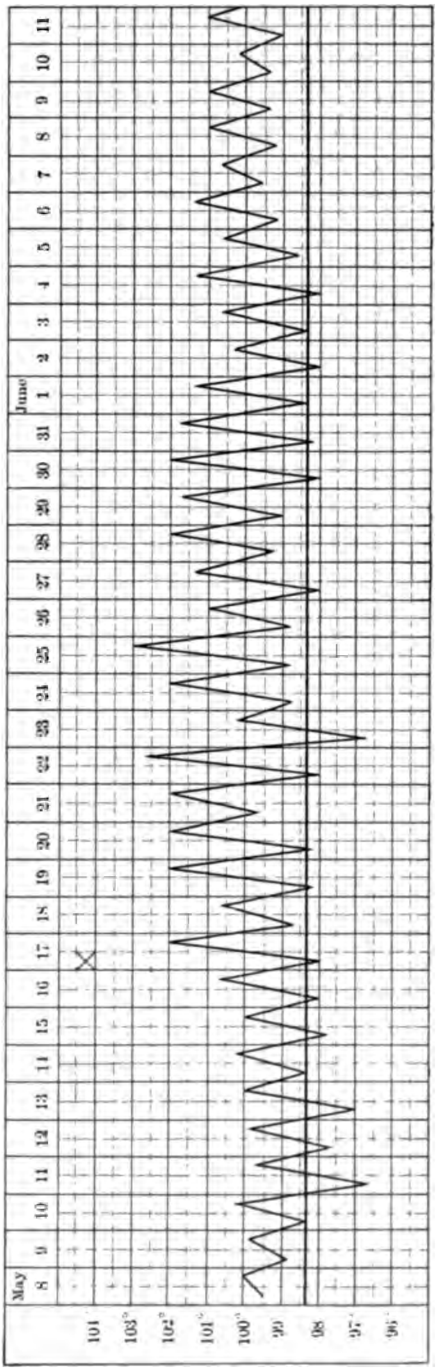


CHART II.—CASE OF ACUTE ENDOCARDITIS (p. 172). Cross indicates time of development of signs of pneumonia in right apex.

strychnine and yeast-nuclein in full doses. The diet was as hearty as his feeble digestion would permit. He felt so comfortable that he was kept in bed with difficulty. Things remained in this condition for two weeks, when he one day complained of localized pain and stiffness in the calf of the left leg, but with no objective phenomena.

His cough was so persistent that repeated examinations of the lungs were made, at first with negative results. At length I discovered moderate dulness of the right upper lobe with ill-defined bronchial breathing, but no râles. About this same time enlargement of the spleen became demonstrable. At a loss to account for the changes at the right apex, I requested Dr. Arthur R. Edwards to see the patient. He agreed in the view that there was probably septicæmia with endocarditis, but could not give a satisfactory explanation of the pathological condition in the lung. The pain in the left leg he thought was due to embolism. The faint blush at the root of the nails with tenderness, pain, and moderate clubbing was also observed by him, but without special comment.

Coincidently with the appearance of distinct signs in the right apex the fever rose a degree or two, headache was complained of, and cough without expectoration became troublesome. This condition, which I now believe was a pneumonia of the same bacterial origin as the general sepsis, persisted for about two weeks, and then gradually disappeared, the lung clearing up, and the patient's general condition returning to what it was before this attack.

For the next month or six weeks the clinical picture remained nearly *in statu quo*. Occasionally he spoke of sudden pains in the shoulder or elsewhere, which lasted for a few days and then vanished. Once he complained of pain and tenderness of the liver, and the organ became slightly more enlarged. This condition persisted for perhaps a week, and then disappeared slowly. All this time there was the same relentless pyrexia, which at times fluctuated rather more than before—but with exception of its becoming somewhat weaker in action and sounds, the heart showed no distinct change.

The character of the temperature is shown in the annexed chart. Credé's ointment was rubbed into the skin freely for two weeks, and was then succeeded by daily injections beneath the integument of an old antistreptococcus serum in doses of 10 cubic centimetres. Although kept up for more than a week this treatment produced no results, unless perhaps a feeling of somewhat greater strength and a slight reduction of temperature.

At length, near the middle of July, the patient, who all the time declared he did not feel very ill, suddenly experienced an excruciating pain in the third finger of the left hand. The finger became cold, pale, and exquisitely sensitive to touch, as well as so painful that it prevented sleep. Examination made it apparent that an embolus had lodged in the artery near the middle of the third phalanx. Plugging was so complete and the patient's suffering so intense that it became necessary to amputate the finger on the fourth day. It was now apparent that he was failing steadily, although slowly. The heart-rate increased to 120, the apex-impulse less defined, the first sound inaudible, but the murmur not appreciably more distinct, indeed rather less intense, and the fever increased, but without rigors or sweatings. Indeed, throughout the illness these had been conspicuous by their absence.

I saw him for the last time on the afternoon of July 22d, at which time he declared he felt so comfortable and happy that he believed he was going to get well. His countenance was slightly suffused, and as he had not emaciated greatly, his friends could not realize how ill he in reality was. During the forenoon of the 24th he was suddenly seized with pain in the lungs, which was followed by violent coughing and the expectoration of blood. Evidently pulmonary infarcts had taken place and betokened the near approach of the end. The cough grew so incessant as to require considerable doses of morphine hypodermically for its control. These were followed by unconsciousness, in which condition he lingered until the afternoon of July 25th, when he suddenly expired.

The course of this undoubted case of septic endocarditis occupied five months from the attack of tonsillitis. It was plainly one of sepsis from the date of his visit to my office, yet without the occurrence of embolisms there was little to direct one's attention to the heart. I was familiar with the previous condition of the heart, and hence able to detect slight alteration in the physical signs which might have been otherwise unrecognised. Yet the thing that riveted my attention from the start was the almost unswerving persistence of the pulse at very nearly the same rate, 105, in spite of rest in bed, until near the termination of the case, when it rose to 120.

A highly interesting feature also was the condition of the terminal phalanges of the fingers.' The faint redness, pain, and, it seemed to me, slight increase of heat, were due to capillary dilatation, which caused the ends of the fingers to become distinctly bulbous in the course of a few months. During the preceding winter I had observed this same phenomenon, only to a much more marked extent, in a young woman who was in the ward at Cook County Hospital suffering from mitral disease, and, judging from the symptoms, from endocarditis. She subsequently left the hospital, so that I cannot state the cardiac condition definitely. In her case the finger-ends became distinctly red and unmistakably bulbous. In both these instances there was pyrexia associated with cardiac disease. May not this phenomenon in the finger-tips point to something more than mere capillary paresis, perhaps to such a disturbance of the cardio-vascular apparatus as may suggest involvement of the heart as well as general infection? It is a matter of profound regret that an autopsy could not be obtained in the case which has been narrated at such length.

Physical Signs.—*Inspection* is of very negative value, since although it may enable one to detect signs of circulatory disturbance, it does not furnish proof of such disturbance being due to endocarditis. Besides, valvulitis is so often masked by the symptoms and physical signs of the primary affection that inspection of the patient reveals only such changes in the patient's appearance as belong to the rheumatism or other original disease.

Palpation is likewise of minor aid in the detection of acute endocarditis. It may assist in the recognition of some old-standing valvular lesion, or in determining the presence of abnormal pulsations, variations in the force, position, and extent of the apex-beat, the possible development of thrills in the course of the affection, enlargement or not of the liver, etc., all findings that bear in certain cases on the condition of the heart, but, like inspection, it cannot furnish direct evidence of the existence of acute endocarditis. Its chief value is in the *study of the pulse*, which in all suspected cases should be carefully studied, changes in its tension being often of greater value than alterations of rate or rhythm.

Percussion.—This is indispensable if one will correctly appreciate the significance of auscultatory phenomena, since the condition which occasions an organic murmur also leads to dilatation or hypertrophy, and therefore to a corresponding alteration in the area of cardiac dulness. If endocarditis occasions mitral incompetence, it will also eventually occasion secondary enlargement of the right ventricle. Consequently *daily percussion* over this chamber should be made in order to detect the earliest evidence of any alteration in the outline of the right and lower cardiac border. On the other hand, aortic disease produces increase of dulness on the left side with gradual alteration in the position of the apex-beat. Accordingly percussion is of very great value, since without the information derived from it a careful diagnostician would scarcely venture on an opinion concerning the nature of an endocardial murmur, especially an apex-murmur, which, as is well known, is of frequent occurrence in febrile affections without inflammatory changes in the valves.

Auscultation.—Even this furnishes only exceptionally a trustworthy means of recognising endocarditis, for, as will be shown later on, a murmur is not always to be accepted as an indication of valvulitis.

It is not so much the detection of an actual murmur that is significant, as it is the *recognition of changes* in the character and relative intensity of the cardiac tones. Daily careful study of the sounds must be made therefore. The earliest evidence that all is not right at the mitral orifice (the seat of inflammation in 50 per cent of cases) is said by English observers, who it must be acknowledged have given close attention to this subject, to be not a murmur, but a *muffling* or *indistinctness* of the first sound at the apex. A blowing murmur, which is purely accidental, may accompany the first sound in a given case, and hence the occurrence of a murmur with the sound is not so significant as an alteration in the sound itself, since it is reasonable to assume that if the production of the sound is interfered with it may be by inflammatory changes. Such alteration of the second sound at the base is likewise suspicious of endocarditis affecting the semilunar valves.

If in a case impurity of any of the tones is observed, it is very likely to grow into a more or less distinct murmur in the course of time, while there will also develop the secondary changes in the heart appropriate to the lesion, whatever it may be.

A *diastolic murmur* developing in the aortic area may be set down as not accidental, and therefore *indicative of inflammation* at this ostium. If this inflammation seriously impair the integrity of the valve, it will be shown in time by the occurrence of the secondary cardiac and vascular signs, which are described at length in the chapter on Aortic Regurgitation.

A *presystolic murmur rarely develops* as a result of acute endocarditis unless the process is ingrafted upon an old-standing mitral regurgitation. Inflammation attacking previously healthy valves usually produces incompetence of these, or this combined with a minor degree of obstruction. Therefore, the murmur is apt to be purely systolic, or systolic with a very short, scarcely recognisable presystolic one. When, however, an old endocarditis affecting the mitral valve becomes rekindled, as not infrequently happens, then a presystolic bruit may develop, and so developing it furnishes almost indubitable proof of endocarditis being present. This circumstance has more than once enabled me to diagnosticate correctly the occurrence of endocarditis, as proved by subsequent events.

A systolic murmur in any of the cardiac areas must always be regarded with doubt until its true significance is shown by the development of secondary physical signs. This is true of such a murmur even in the aortic notch, for experience has taught that even here a murmur may be accidental. It is, however, in the case of an apex systolic murmur particularly that caution must be exercised. (1) Because accidental bruits are most often mitral, or mitral and pulmonic. (2) Because such a mitral systolic murmur may be the result of a previously existing regurgitation, and not at all due to the rheumatism in the course of which it may happen to be discovered. (3) Because, as shown by a case reported by James W. Walker, of this city, an endocarditis may exist without its giving rise to any murmur whatever. In his case the anterior and left posterior aortic cusps presented on their surface large masses of soft, friable, nodular vegetations, which had filled the ostium, and yet repeated and careful examinations during life had utterly failed to detect any murmur.

Diagnosis.—These three considerations make very plain the *fallacy of depending upon the presence or absence of a murmur* in making a diagnosis of acute endocarditis. Inasmuch, therefore, as acute simple endocarditis may escape detection entirely or may be only suspected, one is frequently compelled to leave its diagnosis an open question until after it has occasioned sufficient structural change to produce secondary physical signs. One must therefore depend upon the *history* and *symptoms* even more than on distinctive objective signs.

Even the pyrexia, acceleration of the pulse and respiration, præcordial pain, nervousness, and restlessness may all be attributable to the rheumatism. When the endocarditis is mural—i. e., confined to the lining membrane of the cavities—the valves escaping altogether, a positive diagnosis cannot be made without the occurrence of embolic phenomena.

Differential Diagnosis.—Acute pericarditis is scarcely likely to be mistaken for acute simple endocarditis, and yet it is conceivable that such might be the case when the friction-murmur happens to coincide with one of the heart-sounds, and hence simulate an endocardial bruit. In such a case the greater pain and the subsequent development of signs of effusion ought to set one right. Should an inexperienced auscultator mistake a to-and-fro pericardial rub over the base of the aorta for the double murmur of aortic insufficiency of recent origin, he may be able to correctly interpret the rub by noting the absence of the secondary cardiac and vascular signs of an aortic valve-lesion.

Pernicious anæmia may under some circumstances very closely simulate acute endocarditis without embolic phenomena. I have seen such a case, in which the systolic apex-murmur of ever-increasing intensity, the low irregular pyrexia, great prostration, pain in the hepatic region, with nausea and vomiting, were, together with a history of infection of the forefinger and lymphangitis four months earlier, highly suggestive of a low grade of endocardial inflammation, particularly as the patient gave additional history of rheumatism. The blood-examination and ultimate autopsy findings established the correct diagnosis.

The Diagnosis of Ulcerative Endocarditis may in some cases be easy, in others only a matter of conjecture. What has been said concerning the physical signs of the simple applies equally well to the malignant form. In some instances the entire clinical picture is that of general sepsis, and there are no findings to betray its localization in the heart. In others characteristic signs of valvular insufficiency develop or the tokens of an old lesion undergo modification, or indications of a new lesion become added to those of a pre-existing defect. For these reasons repeated and minute examinations of the heart are necessary. The detection of a murmur is in itself of small moment sometimes, but the discovery of *changes in the rhythm* of one already existing, as by a presystolic being prefixed to a systolic bruit, or an alteration in its timbre, a previously soft one becoming harsh or musical, or the addition of a diastolic murmur to a systolic one, all such modifications are of great significance, and should be carefully noted.

The most reliable aid in the diagnosis of malignant endocarditis is found, however, in the history and symptoms. One must always seek for an efficient etiological factor. For example, it was not a great while ago that I was asked to see in consultation a young woman who was running an irregular course of fever, was emaciating, losing strength, and had a cardiac murmur. It was evident at a glance that she was suffering from pronounced septicæmia, but the question that the physician wanted cleared up was whether the cardiac findings indicated septic endocarditis. There was history of an old rheumatic mitral disease. It did not require very long search to find evidences of cutaneous infarcts, and when in response to inquiry concerning a vaginal discharge, the nurse stated that there had been an extremely offensive one earlier in the illness, and when a vaginal examination disclosed cervical laceration, the chain of testimony was complete. An abortion had led to uterine infection, this to septicæmia, and this latter to an ulcerative process within the heart, which was predisposed to inflammation by its old-standing mitral lesion.

Symptoms of pyæmia—i. e., chills, fever, and sweating—indicate general sepsis, but do not warrant the inference that the endocardium is involved. This can only be assumed when embolic phenomena are discovered or there are some cardiac findings as well as symptoms of general sepsis. It is very necessary, therefore, to make daily examination of the skin, spleen, and urine for detection of the changes already mentioned as forming an essential part of the symptomatology of this form of endocarditis.

In the very obscure cases in which embolic phenomena are not present and there are no cardiac findings to explain the pyrexia and other features that point to infection, yet in which the feebleness of pulse and heart-sounds suggest the possibility of a primary focus of infection in the endocardium, information is likely to be obtained from examination of the blood. Usually in infections there is pronounced leucocytosis, but in a few cases Neusser found an absence of increase, and he consequently concluded that when in a given case of septicæmia there is either an absence or possibly a decrease of leucocytosis, it points to the likelihood of malignant endocarditis. Bacteriological examination of the blood in a suspected case is not often productive of results, and yet should be made when all other means of arriving at a diagnosis fail.

Differential Diagnosis.—It is not sufficient to merely make a diagnosis of acute endocarditis; one must also determine its nature. Therefore in making a differential diagnosis the following points may be of assistance: (1) The possible etiological factor; in the simple form, articular rheumatism, chorea, scarlatina, or some other generally recognised cause of benign endocarditis; in the ulcerative, some antecedent pus infection or focus of suppuration, croupous pneumonia, gonorrhœa, rectal abscess, otitis media, quinsy, trauma, furuncle, carbuncle, leg ulcer, etc. (2) The character of the temperature. In simple endocarditis fever may be absent or due to the primary disease, subsiding with the disappearance of the rheumatism, etc., or it may pursue a mild continuous course. In the ulcerative form the temperature-curve is that of pus-infection of the characters that were described in the symptoms. (3) Blood changes and bacteriological examination of the blood are negative in the simple form, while in the malignant there may be pronounced leucocytosis. The occurrence of septic phenomena without such increase points to septic endocarditis. (4) Urinary findings. The discovery of albumin, pus, and blood with casts is in favour of mycotic endocarditis, since hæmorrhagic nephritis is not a part of the clinical history of the simple form. (5) Hæmorrhages into the skin or from the mucous membranes may occur in the ulcerative, but not in the benign variety of endocarditis. (6) Embolic phenomena, although occasionally observed in the simple, are yet generally found in the malignant endocarditis. (7) Enlargement of the spleen is in favour of the mycotic as against the simple form. (8) Recovery is the rule in simple and the exception in ulcerative endocarditis, although its course to a fatal issue may be slow.

Typhoid fever is the disease above all others for which ulcerative endocarditis is likely to be mistaken. As a matter of fact, most cases of the latter affection are diagnosed as enteric fever, and so regarded until in the dead-house they are found otherwise. The points of difference are the following: (1) In typhoid fever the temperature is not so erratic, and in the first week often displays the characteristic gradually ascending curve. (2) The pulse in this disease is usually slow, out of proportion to the degree of temperature. In its want of tension during the height of the malady it may be like that of endocarditis. (3) Splenic enlargement comes at an earlier period in enteric fever, and is more considerable. (4) Rose-spots usually appear between the eighth and twelfth day, are apt to be in crops upon the trunk, disappear temporarily on pressure, are of a nearly uniform size, and have a darker centre, fading out towards the periphery. Cutaneous embolisms, on the contrary, appear most often upon the extremities, are irregularly distributed, of variable size, do not fade on pressure, and have a necrotic pale centre, becoming of a deeper colour towards the edge. (5) The stools of abdominal typhus are often, though by no means always, diarrhœal, have the pea-soup appearance, and contain the Eberth bacillus. (6) Typhoid-fever patients are very apt to manifest signs of bronchitis. (7) Excepting epistaxis early in its course and possible hæmorrhage from the bowel during the middle or latter portion, hæmorrhages are not common in typhoid fever. (8) In the Widal agglutination test we now possess a reliable means of differentiating enteric fever from malignant endocarditis, and it should invariably be made in every doubtful case.*

The value of this differential test was shown in a case under my care a few months ago. A young man with extreme aortic regurgitation of rheumatic origin was under treatment for attacks of præcordial pain, and as there were symptoms pointing to a rupture of compensation he was put to bed. A few days thereafter he began to manifest a low grade of irregular temperature, but without any other symptoms. His pulse remained disproportionately slow, and I at once suspected typhoid fever, although not unmindful of the possibility of endocarditis. Rose-spots were never discovered, notwithstanding repeated daily inspection of the trunk, and splenic enlargement could never be satisfactorily made out either by palpation or percussion. There was no diarrhœa at any time, the bowels being rather confined. The Widal test was resorted to at the end of the first week, and settled the diagnosis as one of enteric fever. Had this means of differential diagnosis not been available I should have felt exceedingly uneasy as to the nature of the case and its possible outcome. As it was, I felt no more anxiety than would be natural in such a severe valvular lesion, complicated by the occurrence of the abdominal typhus, and a possible infection of the chronic endocarditis in consequence.

---

* According to MacFarland, there was in 4,000 cases only 4 per cent of error.

The error of mistaking ulcerative endocarditis for malarial infection is so easily avoidable nowadays by the discovery of the plasmodia that it ought never to be committed, and is inexcusable.

Prognosis.—This depends not only upon the nature of the endocarditis, whether benign or ulcerative, but also upon certain modifying conditions, as the extent and seat of the inflammation, the concurrence or not of acute pericarditis or myocarditis, whether it is a first attack or has been ingrafted upon a chronic endocarditis, and lastly upon the presence or absence of septic infarcts. If the inflammation expend its energies in a local deforming process through the development of new connective tissue within the valves, or the formation of vegetations upon their surface, or that of the contiguous orifice, the endocarditis does not usually destroy life directly, but leaves the individual with a permanent valvular defect. This statement must be modified, however, in accordance with the seat of the inflammation. If this is confined to the left auriculo-ventricular opening, which fortunately is the case in at least one-half of the instances, the immediate prognosis is much less grave than when the endocarditis attacks the aortic valves, rendering them incompetent. Rapidly induced insufficiency occasions dilatation of that chamber into which the blood regurgitates.

The secondary effect of endocarditis of the *mitral* cusps is dilatation of the left auricle, of the *aortic* cusps, dilatation of the left ventricle, and there is abundant proof, both clinical and otherwise, that dilatation of the auricle is less dangerous to life than dilatation of the ventricle. Moreover, in mitral regurgitation, the resistance offered by the walls of the left auricle is re-enforced by the column of blood in the pulmonary vessels and by the right ventricle, while in insufficiency of the aortic valve there is not only danger of paralysis of the left ventricle from overdistention, but if in consequence of stretching of this cavity and of the left auriculo-ventricular ring the mitral valves become relatively incompetent, the evils and dangers of mitral are added to those of aortic regurgitation.

If the inflammatory process extend also to the myocardium or pericardium the prognosis becomes far more serious, since the myocarditis favours a *rapid dilatation* of the organ. Sturges directed attention to the liability in children to inflammation of all of these structures, giving it the name "acute carditis," and pointed out the extremely serious nature of this condition. The gravity of the prognosis in these cases is attested by the following figures, to which reference has already been made in the chapter on Acute Pericarditis: Of 150 cases of fatal rheumatic endocarditis in children, Poynton found the pericardium healthy in only 9, while in 34 the myocardium showed changes of one kind or another. Death was thought attributable to the condition of the myocardium rather than to that of the endocardium.

If an acute endocarditis becomes ingrafted upon the chronic process, or attacks valves already the seat of sclerotic changes, the prognosis becomes very doubtful, since it is a well-known fact that under such circumstances the inflammatory process is very likely to prove septic. Moreover, even if the endocarditis should not be malignant, it is certain to intensify the changes already existing, either by causing still greater destruction of the valves or by transforming a predominating insufficiency into a stenosis through the development of thrombi about the edges of the opening. Thus a lesion which was compensated may be converted into one of such gravity that compensation is never again possible.

The occurrence of embolisms renders the prognosis exceedingly serious, both as to the immediate and remote effects. Even in simple rheumatic endocarditis an embolus may be carried into the brain, the left *middle cerebral artery* being the one most frequently plugged, and occasion hemiplegia. In the case of endocarditis of the right heart, pulmonary infarcts frequently contribute to the fatal termination. Should the emboli be septic, more than a mechanical effect is produced. Single or multiple abscesses in the spleen, liver, kidneys, or even scattered throughout the body, set up symptoms of general infection. These are the cases properly classed under the category of malignant endocarditis, since in them death is the inevitable result. Should the urine at any time display the characters of hæmorrhagic nephritis, it is to be regarded as an omen of evil import.

The very interesting and practical question arises, Can acute rheumatic endocarditis subside, leaving the valves uninjured? This query has been answered in the affirmative by some writers, their belief being based upon the disappearance of a systolic apex-murmur that had been observed to develop during an acute rheumatic attack. My experience has been too limited to warrant my forming an opinion upon the subject, yet I frankly state I would be loath to accept any other than post-mortem evidence of the correctness of such a belief. Under the influence of infection and pyrexia, weakening of the myocardium and papillary muscles may very readily occasion dilatation, and a systolic murmur in the mitral area be developed. With returning health these may regain their tone, and the dilatation and murmur disappear. Can any one assert therefore, without fear of contradiction, that the appearance and subsequent disappearance of such physical signs indicate recovery from acute endocarditis, and not from cardiac dilatation? The following case observed during convalescence from pneumonia is one in point: A healthy young married man of twenty-seven passed through a pneumonia of the right lower lobe in the fall of 1899. About a week after the crisis, when convalescence was progressing finely, he arose from bed early one morning and walked into the adjoining bath-room to pass his urine. He suddenly became weak and dizzy, and upon returning to his bedchamber his pulse was observed to be 135 to the minute and weak. Whenever during that day he attempted to walk about the room his pulse immediately arose in frequency and became correspondingly diminished in strength. I was asked to see him that same evening, and found him reclining on the sofa, his pulse about 100, regular, but compressible, the apex-beat feeble, in the fifth left interspace slightly outside the nipple-line.

Relative cardiac dulness was increased transversely, particularly to the left, reaching 12.5 centimetres to the left of the median line. The temperature was normal, respirations tranquil, and the patient had no sense of dyspnœa. Cardiac sounds were everywhere audible, but the aortic second was weak, and accompanying the first sound at the apex was a faint systolic blowing murmur. There was no history of previous attacks of rheumatism, and until the date of his pneumonia the patient had indulged in much athletic exercise without shortness of breath or palpitation. Realizing the possibility of an acute endocarditis of pneumococcus origin, I insisted upon absolute physical repose, ordered light diet, and a gentle saline aperient.

On the following day, the condition being essentially the same, $\frac{1}{40}$ of a grain of strychnine sulphate three times a day was ordered. As the temperature remained normal and the murmur had not increased, two days later tincture of digitalis was cautiously administered. Within twenty-four hours the left ventricle had come down 0.5 centimetres, and upon the digitalis being increased, the next twenty-four hours witnessed a still further diminution in the extent of relative cardiac dulness to the left. In the course of the next week or ten days the heart measured but 10 centimetres to the left of the median line, and was normal at the right (Fig. 32).

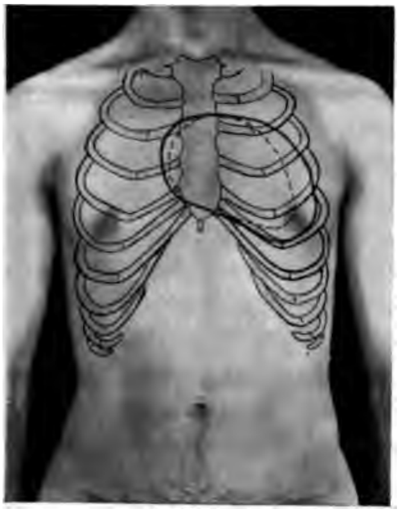


FIG. 32.—DIMINUTION OF RELATIVE CARDIAC DULNESS IN ONE WEEK, UNDER TREATMENT. CASE (p. 185).

Two months subsequently, after the patient had been without medicine for several weeks; and had returned to his usual mode of life, the left ventricle measured but 9 centimetres, a reduction of more than 3 centimetres since the date of my first examination. Was this case to be regarded as one of acute endocarditis following croupous pneumonia? Certainly not. It was one of simple acute dilatation, chiefly of the left ventricle, resulting primarily from asthenia of the heart-muscle in consequence of the effect of the toxins of the pneumococcus.

Treatment.—Clinical experience the world over accords with the conclusion naturally drawn from a consideration of the pathology and morbid anatomy of acute endocarditis—viz., that when the process has once become established, *we possess no means* of causing absorption of inflammatory product or restoring the endocardium to a healthy state. It should be our aim, therefore, to prevent where we cannot cure. Our first duty is to study the efficacy of prophylactic measures. Our efforts in this direction should not be restricted to prevention of endocarditis, but should first be directed against that disease, articular rheumatism, which is so largely responsible for inflammation of the cardiac structures. Proper sanitation and attention to the diet, clothing, habits, and occupation of patients may do much to this end.

Of special value are all measures calculated to maintain a high standard of nutrition, and persons of distinct rheumatic diathesis should be impressed with the danger of exposure to wet and cold. Children in whom rheumatic manifestations are obscure, should be carefully examined whenever ailing, for possible evidence of rheumatic infection, and if this be discovered, should promptly be given some preparation of a salicylic acid.

Much has been written concerning the prevention of cardiac involvement during rheumatic attacks; and when the salicylic-acid treatment of rheumatism came into use, strong hope was entertained of its ability to prevent endocarditis. Even now there are those who believe that by diminishing the severity of, or even cutting short, the rheumatic attack, this treatment lessens the liability to cardiac inflammation. The same also may be said of the alkaline treatment, or of the combinations of the alkalies and salicylates. For the most part, observers of long experience have come to the conclusion that whereas the salicylate-treatment does not assure the prevention of endocarditis it would better be persevered with, since if properly administered it is not likely to do harm. For my part I do not believe in the prophylactic power of this drug over the cardiac manifestations of articular rheumatism.

Given a case of this disease in which salicylate of soda is employed, and acute endocarditis or pericarditis does not develop, can any one assert it would have occurred had the remedy not been administered? Are there any statistics to show that endocarditis has been less frequent than before the use of the salicylates? Even if one or more series of rheumatic cases treated with this remedy show a smaller percentage of endocarditis than others not so treated, how can one be sure that the difference in results is not purely accidental, since all rheumatic attacks do not inevitably lead to cardiac inflammation?

By all means during a rheumatic attack resort to salicylic acid, or one of its salts, to potash or soda, local applications to the affected joints, to regulation of the diet, and any other approved means of antirheumatic treatment. But do not be too confident that endocarditis will not develop. Should it not, consider yourself and the patient fortunate.

I confess to the same scepticism concerning the efficacy of local treatment of the præcordium, as leeches, blisters, and cold applications, in preventing acute endocardial inflammation. The only prophylactic measure that appeals to me as rational is the procurement of as much *rest to the heart* as possible, by keeping the patient quiet during his attack of rheumatism, that the valves may not suffer *trauma by reason of strain.* Fortunately, in an acute attack of severity the urgency of the symptoms compels the patient to remain at rest; but in cases of subacute rheumatism, particularly if an old valvular defect already exists, the patient should be urged to remain at rest, so as to lessen the tension of the valves and the possibility of having inflammation rekindled in them. This is often irksome to the patient, but if he has the reason explained to him he is likely to acquiesce, although perhaps with no very good grace. Even after all the rheumatic symptoms have disappeared the patient should be cautioned against violent exercise, and should be kept under rather frequent observation, that the earliest evidence of endocarditis, should such arise, may not be overlooked.

Upon the occurrence of acute endocarditis, or of subjective or objective symptoms suspicious of such an inflammation, the patient should promptly be put to bed, if not there already, and kept there as quiet as possible, both mentally and physically. The object of this is apparent; bodily exertion as well as mental excitement augments the frequency of cardiac contractions and subjects the valve-curtains to increased strain. *The same principles should apply to the treatment of inflamed valves as to that of inflamed joints.* The use of the latter not only causes pain, but intensifies the inflammation. Unfortunately, the heart cannot be put at entire rest, but by slowing its contractions its diastole or period of rest is lengthened and its contractions are less violent. Theoretically, at least, the inflammatory process would thus be less active and the danger lessened of rupture of the inflamed and tender cusps, or of dislodgment of a soft, not firmly seated thrombus, and the formation of embolism. If the heart's action is violent or too rapid, attempt should be made to quiet it by placing ice-bag to the præcordium or by the administration of bromides.

Digitalis is very commonly administered for this purpose, but it cannot be stated too emphatically *that this drug is inadmissible in the treatment of acute endocarditis.* It not only does no good, but is positively harmful. Although capable of slowing the heart, digitalis at the same time increases the strength of systole, and thereby subjects the valves to more than ordinary strain. The benefit to be derived from a slowing of the contractions is offset by the injury to the valves and by other dangers possible from this more forcible closure, as already explained. It is better to let the heart keep its own gait than attempt to control it by possibly injurious means.

Aconite or veratrum viride are likewise injurious, but in a different way. They are depressors to the myocardium; and if this be inflamed or weakened by serous infiltration, there is danger of these drugs causing serious dilatation. The same objection cannot be urged against the local employment of cold, and as a matter of fact this therapeutic agent is highly praised by those who have given it an extended trial. As stated in the chapter on Acute Pericarditis, the ice-bag is preferable to cloths wrung out in ice-water, since they do not subject the patient to the danger of taking cold by wetting the clothing, and for the same reason are more comfortable. The ice-bag should not be applied directly to the bare skin, but a dry, light cloth should be interposed. Should the heart and circulation have become very feeble, cold had better not be resorted to, because cardiac depressors are no longer indicated.

Hot applications to the præcordium are then more serviceable on account of the stimulating effect they produce. Vesication of the præcordium, either in the form of one large blister, or of repeated small blisters, is a treatment that once met with much favour, but is objectionable, since it occasions more nervous irritation than it is likely to do good. The application of mustard or the tincture of iodine or of a turpentine stupe are less objectionable because less severe, and are sometimes capable of alleviating pain.

Mercurials and tartar emetics are now known to exercise no restraining influence over the inflammatory process, and are therefore no longer used by the best authorities. Moderate doses of iodide of potash have been recommended, in the hope of restricting the formation or promoting the absorption of the inflammatory products. It is, however, doubtful if this remedy possesses any such influence in the course of acute endocarditis.

When medicines are powerless to cut short an attack, or even probably to diminish its severity, we are left to a purely symptomatic treatment. Pain and restlessness should be alleviated by the use of opium, and in the case of adults morphine hypodermically is the best mode of administration. In children great care must be exercised in its use, and it is always well to first try the efficacy of bromides in conjunction with cold applications and soothing liniments. Antipyrine, phenacetine, and other remedies of this class are capable of exerting depression, and if employed at all should be in small doses and with strychnine or some stimulant.

The pyrexia of acute simple endocarditis is usually not high, and therefore such antipyretics are not likely to be needed for the reduction of temperature. If this should become necessary, it would be best attempted by judicious sponging. Insomnia may be prevented by bromide, paraldehyde, sulphonal, or trional, or, best of all, by some preparation of opium.

So soon as the endocardium is found to be the seat of acute inflammation the physician should constantly bear in mind the possibility of the heart finally succumbing through weakness, if not structural change of the myocardium. The organ should be sustained, therefore, by that best of all heart-tonics, strychnine. Opium is also a heart-tonic, and while being given for the relief of pain, also supports the heart, provided it be not administered with greater frequency or in larger doses than are required to alleviate the symptoms. Sulphate of strychnine is, however, the remedy on which chief reliance should be placed. Given in moderate doses, at first $\frac{1}{60}$ of a grain to an adult three times a day, it may be increased to $\frac{1}{30}$, or even to $\frac{1}{20}$, if signs of myocardial weakness supervene. Should the disease assume a grave character, and attacks of threatening asystolism make their appearance, by cyanosis, feeble and irregular pulse, paroxysms of dyspnœa, or signs of pulmonary œdema, the heart should be promptly stimulated by ammonia, camphor, ether, brandy, and the like. Inhalations of oxygen may also be resorted to, and are likely to prove temporarily, if not permanently, beneficial.

The patient's general strength should likewise be sustained by nutritious, though light, diet. Milk, beef juice, an occasional raw egg, soup, broth, and wine jelly are all serviceable. A cup of soup (prepared from Mosquera's beef jelly), tropon and somatose, form admirable adjuvants to the dietary. The nourishment should be given frequently, every two to three hours, and in small amounts, care being taken to avoid all articles of diet which occasion gaseous distention of the stomach and intestines. Constipation should not be permitted, and even if the action of the bowels is regular, benefit is likely to accrue from the occasional administration of a blue pill or small dose of calomel, followed by a gentle saline aperient. The urine should be watched, and if it become bloody or albuminous the diet should be restricted to milk, and the patient urged to drink freely of pure water.

Acute Ulcerative Endocarditis.—Fortunately there are gradations in the severity of this type of the affection; were it not so the physician would be but a helpless spectator of the ravages of this dreadful malady. Indeed, such is his attitude in severe cases, or in those that have made considerable progress before recognition of their true nature. The first duty of the medical attendant is to *search for the cause*—that is, the source of the primary infection—and, if this is discovered and can be removed by surgical interference, to promptly resort to such treatment. This, alas! is not generally possible; but if, as Sir Douglas Powell thinks, unsanitary environment and exposure to sewer-gas emanations are capable of setting up fresh infection in a case of old-standing valvular disease, then the patient should be promptly removed to a healthful location.

The next indication is to resort to every means which affords any hope of re-enforcing tissue resistance, as a nutritious and easily assimilated diet. The lines of treatment already laid down for the sustaining of the heart and protecting it against the injury resulting from unnecessary work in the simple form are equally applicable to the ulcerative. Indeed, they are still more urgently required; for not only is inflammation more destructive and likely to invade the myocardium, but even when this escapes ulceration or abscess formation the heart-muscle is likely to suffer from the enfeebling effect of the toxæmia.

In severe cases the prostration of the patient generally calls for the administration of small, frequently repeated doses of brandy or ammonia. The use of alcohol in conditions of sepsis is very generally employed, and, in the opinion of many clinicians, is highly useful. Some indeed advocate whisky in large and frequently administered amounts. Strychnine should be given in full doses, and will yield the best results if administered hypodermically. Quinine was formerly exhibited in doses of 15, 20, or more grains for the control of the fever; but with a clearer knowledge of the etiology and pathology of this affection, we now know that this remedy can exert no controlling influence over its course. Iron and arsenic have also been employed, and Powell speaks highly of the latter, not as a curative agent, but simply as a cardiac and general tonic.

Attempts have been made to introduce into the system antiseptics in sufficient quantity to exert at least a modifying influence upon the sepsis. The one deserving special mention is sulphocarbolate of soda, which has been thought in mild cases to exert a favourable influence. Sansom has reported one case in which during its use such improvement took place that the patient left the hospital; she returned, however, and succumbed to a fresh attack or accession at the end of ten months. "At the autopsy the diagnosis of septic endocarditis was confirmed, the mitral, tricuspid, and aortic valves being diseased and infiltrated with micrococci."

Dreehfeld, in speaking of this remedy in ½-drachm doses, mentions a case reported by Sansom (probably the one just quoted) in which, when death took place at a later period, "distinct cicatricial tissue was found at the site of the old ulcerations."

If this or any other antiseptic remedy, as salol and salophen, is to do good, it must be in very large, frequently repeated doses, so as to rapidly bring the system under their influence, and should then be continued for a considerable time. These latter remedies recommend themselves in cases with a rheumatic element, because composed of salicylic as well as carbolic acid; but the depressing effect of the former upon the myocardium must not be forgotten. For my part I am inclined to attribute whatever benefit has seemed to follow such treatment to their local antiseptic action upon the intestinal tract. Fermentative processes and diarrhœa, as shown by fœtor of the discharges, are very common within the digestive tube of patients suffering from sepsis. Such a condition may not only intensify the pyrexia and other symptoms of infection, by itself setting up an infection of intestinal origin, but it prevents the proper digestion and assimilation of nourishment. If now this putrefactive fermentation can be prevented by intestinal antisepsis, the patient's nutrition will improve and his tissue resistance be augmented. It is possible, perhaps, by having this additional enemy thus removed, the system may be able to cope successfully with the primary invader. At all events, the physician should employ these and every other means that afford possible chance of improvement in dealing with so formidable an adversary.

The universal success of the antitoxin treatment of diphtheria, and the encouraging reports that have come from the use of *anti-streptococcus serum* in some cases of pyæmia and puerperal septicæmia, indicate the dawn of a new era in therapeutics. It is to be hoped that in the not very distant future we shall possess a serum potent against each kind of pus-producing microbe. At present we are limited to the serum just mentioned; and inasmuch as the streptococcus is the agent frequently at work in cases of septicæmia, and the judicious employment of this serum appears not to be injurious, we are certainly warranted in giving it a trial in cases of septic endocarditis. This has already been done, although to what extent I am not able to say, nor have I been able to find how many cases of this disease treated in this manner have appeared in the literature to date.

Douglas Powell has tabulated 14 cases of ulcerative endocarditis in which antistreptococcus serum has been employed in London. The results are as follows: Three recoveries, 9 deaths, and 2 in which no favourable result ensued. Powell is of the opinion that these results appear more discouraging than they really are, from the fact that the serum was employed in the late stages of the disease, owing to a natural hesitancy to try a new remedy, and after "large embolic detachments had set up fresh centres of cultivation in many positions." He concludes therefore: "It may be laid down as a principle, governing treatment by this particular serum, that the more distinct the history of a previous endocardial lesion and a subsequent exposure to an infection through a suppurative medium, or a sewer-gas sepsis, the more appropriate the case for the treatment. This rule would discourage its employment in cases in which the pneumococcus, gonococcus, or some other microbes divergent in character from the streptococci and staphylococci were concerned; and if with the recognition of this principle, and its earlier and bolder carrying out, more encouraging results are obtained, it will certainly follow that analogous measures will be found for the circumvention of the other forms of microbic action."

If the primary source of infection, an abscess for instance, be not discovered, and therefore not removed by the surgeon, or if fresh emboli laden with pus cocci repeatedly discharge into various parts of the system, to maintain the already existing sepsis or set up fresh centres of infection, then assuredly antistreptococcus serum will prove of little or no benefit.

If, on the contrary, the patient is suffering from pyæmia, the original portal of infection having been closed, and no fresh intoxication having taken place, then this serum would be of service, even though the streptococcus be not the only microbe concerned in the process. With this formidable streptococcus disposed of, the system ought, theoretically at least, to be able to cope successfully with the other kind of bacteria. Of course, hope of recovery or even improvement can only be entertained in comparatively mild cases, or when, as Powell says, the disease is recognised and treatment begun early. A process with a pronounced destructive tendency cannot probably be checked, but there are cases of septic endocarditis which are shown by the clinical history to be not thus rapidly destructive or malignant. Since no one can foresee how virulent the endocarditis is to prove, the patient should be given the benefit of a doubt, and the serum tried. Gibson suggests that in every case an examination of the blood should be made for possible detection by culture, inoculation, experiment, or otherwise of the nature of the infective agent; but their detection, it must be remembered, is extremely unlikely.

Endocardial inflammation following pneumonia, or in which the pneumococcus has been identified, promises no hope of improvement from this treatment. It is to be hoped that we shall possess some day an efficient antipneumococcus serum, and indeed the researches of the Klemperer brothers and others afford some promise of this being attained.

Personally I have had but little experience with antistreptococcus serum in acute endocarditis. The wife of a physician had suffered for years from an aortic regurgitation of rheumatic origin. At the time I saw her she had been ill for several weeks with moderate fever of a remittent type that fluctuated between about 100° and 102° F., or a little more. She complained much of præcordial distress and paroxysms of pain, also of pain in the lower extremities about the joints, although the latter were not appreciably swollen or tender. The usual antirheumatic remedies—salicylates, alkalies, etc.—did not appear to exert any influence over the affection, and as I believed she was suffering from fresh endocarditis, possibly of a septic type, I advised a trial of antistreptococcus serum. This was obtained from St. Louis, and was given in two doses of 10 cubic centimetres each. Her husband subsequently sent me a report, from which the following has been extracted:

"Mrs. B. had been very sick about one month when you saw her. The attack set in with a spell of tachycardia, lasting between three and four days, pulse-rate near 200 during all that period. The joints were only slightly inflamed, temperature about 102° F., with but slight variation. I gave two doses of antistreptococcus serum three days apart, as you directed, without immediate effect on temperature or symptoms. At end of two weeks, however, temperature subsided nearly to normal. A very heavy erythematous rash followed the use of the serum. She gradually crept from her perilous condition, dropsy disappeared, appetite returned with a fair degree of strength. She had a good deal of bronchitis, and was much worse after you saw her than she was then. No one who saw her at her worst thought she could possibly recover."

The nature of this case was very doubtful, and from Dr. B.'s report the serum appears to have been of doubtful utility. Yet I recall distinctly having subsequently met another practitioner, who had been present at the time of my examination, and who stated in no unequivocal terms that in his opinion the serum had been of benefit.

About a year ago I saw in consultation with Dr. Lovewell a man of about forty who had been ill for a number of weeks with an intermittent fever, rigors, and sweatings, symptoms of cardiac disease, and distinct evidence of an aortic valve-affection, which had not existed before his illness. The origin of the infection could not be ascertained. There were well-marked signs of aortic insufficiency, which from the general septic phenomena and albuminuria could not have been other than a malignant endocarditis. As everything in the line of antiseptics had been tried to no purpose, I advised the use of antistreptococcus serum. The patient survived a number of weeks longer, but died suddenly as a result apparently of emotional excitement. I did not see the patient again, but had news of his condition from Dr. Lovewell, who stated more than once that under the use of the serum the temperature became lower, less irregular, and the other indications of sepsis less pronounced. In fact, the general condition improved so much that the doctor at one time began to entertain the hope of his patient's ultimate recovery.

It will be remembered that in the case reported of my patient of forty, who died of pulmonary infarcts, this serum was likewise employed. It failed to exert any other effect than to slightly reduce temperature and produce a feeling of somewhat greater strength. In this instance it was not used until late in the illness and after embolic phenomena had more than once appeared. I regret that the treatment with the serum was not begun earlier, although I am very doubtful if it would have materially affected the ultimate result. These experiences are too limited to be of value in forming an estimate of the utility of the serum, but inasmuch as its use was not attended by unpleasant effects

I shall certainly continue to give it a trial whenever this seems indicated.

Such cases are so desperate, and the prospect of recovery so slight, that I believe one is justified in resorting to whatever affords even a chance of benefit; and if an old preparation is employed, there is not much danger of producing erythema or articular inflammation, and the remedy cannot prove more harmful than the disease itself, unchecked.

J. Michell Clarke has reported a case of a woman of twenty-two who had had an attack of rheumatism at eighteen, followed by left-sided pleurisy with effusion. She complained of weakness, dyspnœa, præcordial pain, and œdema of the ankles. While under treatment for these symptoms she had a sudden chill, followed by a temperature of 103° F. After remaining high for four days the temperature fell to normal, and after so remaining for about a week, again rose, and prevailed for nine days with a very irregular course. There was a systolic apex-murmur, another loud systolic murmur in the pulmonary area, and a faint diastolic one at the right base. Bacteriologic examination of the blood from a vein was negative. A diagnosis of ulcerative endocarditis was made, and treatment with antistreptococcus serum was instituted. Injections were given from December 31, 1899, to February 9, 1900, sometimes daily, at other times every other day, and once five days intervened between injections. The doses varied from 10 cubic centimetres to 20 cubic centimetres, though as a rule 15 cubic centimetres were given. The patient recovered, and examination revealed the apex in the fifth interspace nipple-line with a loud, blowing murmur throughout the præcordium and posteriorly, but loudest in the aortic area.

Douglas Powell speaks of the administration of yeast, and reports a case in which recovery appeared to be due to this remedy. It is probable that the efficacy of yeast is due to the nuclein of the yeast-cell, therefore in Vaughan's yeast-nuclein we possess a preparation more efficient than a solution of yeast. This may be administered either by the mouth or rectum, a solution, No. 2, being specially prepared for this purpose, or solution No. 1 may be injected under the skin up to 60 or 80 minims in the course of the day. Nuclein or nucleinic acid acts by increasing the number of the polynuclear leucocytes, which are the forms chiefly increased in the leucocytosis observed in infection, and by the increase of which the germicidal action of the blood is augmented.

Many encouraging reports have been made of the favourable effects of yeast-nuclein in cases of pus-infection and cryptogenetic infection. I employed it in one case of acute endocarditis supervening upon an old valvular lesion, which followed a follicular tonsillitis, that may have been rheumatic, but if so was the only manifestation of rheumatism. The remedy was administered by the rectum, owing to the patient's dread of hypodermic injections. Under its influence, or at least during its administration, the mild pyrexia which had existed for about ten days, without showing indication of subsiding, gradually sank to normal. The patient subsequently died. From the foregoing it is evident that the most the physician can do in the treatment of acute endocarditis is to aid nature by helping to maintain the vital powers and by removing obstacles that lie in nature's way.

CHAPTER V

CHRONIC ENDOCARDITIS

Morbid Anatomy.—Two forms of chronic endocarditis are found, one the result of the proliferative processes following an acute inflammation, and the other a part of a general fibroid transformation of the vascular system, arteriosclerosis.

In the form following the acute disease the development of fibrous tissue begins with the organization of the vegetations and thrombi that have formed in the earlier stages. As a rule the vegetations are for the most part absorbed, but the process of organization leaves a slight nodular thickening on the surface of the endocardium. The formation of new connective tissue goes much further than the mere repair of the acute lesions, however, for what reason we cannot say, and the entire substance of the valve is infiltrated by fibrous tissue, which in the course of time undergoes contraction that causes a thickening and deformity of the valve-cusps. This process, then, though initially of an inflammatory nature, eventuates in a sclerosis.

The second form is of *sclerotic origin* from the beginning, and is usually associated with a similar process in the blood-vessels, particularly the arteries. In this process the aortic valve is the one most frequently involved, and the process seems to be often a direct extension of the disease from the aorta. It is, however, by no means rare to find the mitral valve involved, and often both are affected together.

The stiffening and deformity of the valve-leaflets leads to disturbance of their function in two ways: The segments may be retracted or their edges curled in such a way as to permit the passage of blood in the wrong direction (Regurgitation). The condition is then spoken of as insufficiency, incompetence, or regurgitation. If, however, the deformity of the valve is of such a nature as to cause a narrowing of the orifice, the condition is known as stenosis.

Stenosis may be brought about by thickening and rigidity of the valve-segments so that they cannot open perfectly for the passage of the blood, or the remains of vegetations or thrombi, which have undergone organization or calcification, may encroach on the opening. The special ways in which these lesions are produced will be considered in detail under the head of the individual valvular diseases. It should be noted here, however, that stenosis of an ostium and incompetency of the corresponding valve are usually associated conditions, though as a rule one or the other predominates and gives its character to the lesion.

Fibroid thickening of the mural endocardium is not uncommon in connection with chronic valvulitis, especially of the sclerotic type. It may also occur as a part of an interstitial myocarditis. The membrane is thickened and of an opaque whitish or yellowish colour—the latter when fatty change is prominent. Mural endocarditis is often associated with dilatation of a heart-cavity, and is then probably due to the stretching of the membrane.

The *secondary changes* in chronic valvulitis are mainly those due to the circulatory disturbance occasioned by the stenosis or incompetence, as the case may be. If a valve is incompetent it permits regurgitation into the chamber behind during its diastole, and this chamber then receives blood from two sources, the normal one, and through the incompetent valve. Such an oversupply of blood leads to an overdistention of the chamber, and to an increased effort in order to completely empty itself. The continuance of these conditions leads to a permanent increase in the capacity of the chamber, while the increased work thrown on the musculature of the wall causes an increase in its strength and thickness (Hypertrophy).

If the deforming process results in stenosis, the chamber *behind the defect* experiences increased difficulty in expelling its contents, and develops hypertrophy of the kind known as concentric, because associated with little or no dilatation. The chamber in front of a stenosed orifice, on the other hand, is apt to become atrophied and reduced in size, since it receives a diminished supply of blood, and its work is correspondingly lessened.

The disturbances of circulation secondary to valvular disease are by no means limited to the heart itself, but affect the various organs and tissues of the body. The blood-supply to the arteries is lessened, obstruction to discharge of blood from the veins exists, and thus is induced passive congestion, which affects not only the organs drained by the veins, but in well-marked cases also the arterial system which supplies them. In the course of time this congestion reacts injuriously on the heart in a manner to be further elaborated under the head of the respective valve-lesions.

Acute endocarditis is often found associated with the chronic, and indeed the latter predisposes markedly to the former. Changes in the myocardium are also frequent, usually in consequence of nutritional disturbance, which is secondary to the dilatation and hypertrophy, or to associated atheroma of the coronary arteries. Pericarditis is also not infrequently associated with chronic endocarditis, and is generally of the adhesive variety. This is, of course, due to the two diseases having had the same remote origin.

Etiology.—The strictly sclerotic form of endocarditis is not of microbic origin, but is either an expression of nutritional change incident to age, gout, renal and vascular disease, or is the result of strain. That some individuals evince a family tendency to sclerotic changes in the entire circulatory apparatus, as well as in the kidneys, appears proved by the frequent observation of atheromatous valvular disease in two or more members of the same family.

Age is thought to be a factor in the causation of this form of chronic valvular disease; and yet the occurrence of the disease in some individuals at a comparatively early age indicates that there is some other influence at work besides senility.

Chronic endocarditis is so frequently observed in persons of a distinctly arthritic habit that gout has come to be regarded as an important etiological element. With respect to such gouty influence, it seems to me that it is rather the entire manner of living which has to be taken into account. For example, I recently examined a physician's father, whose case illustrates what I mean very well.

Dr. W., from the interior of Illinois, brought his father to me with the following history: The patient was a German, sixty-nine years of age, who had enjoyed robust health up to two years before, at which time he developed redness and swelling, with some pain of the great-toe joints. This was regarded as gouty, and under appropriate therapeutic and dietetic management disappeared. Six months before his visit to me he began to complain of shortness of breath upon exertion, whereupon his son made an examination of the heart and detected a murmur. The routine treatment with digitalis, strychnine, nitroglycerine, and cathartics had failed to produce appreciable benefit, and twice there had been expectoration of bloody sputum. During the previous two weeks he had had two nocturnal attacks of dyspnœa that came on in the small hours, while still a third took place after an evening meal. The son furthermore stated what was of special interest from an etiological standpoint—viz., that his father had always led a sedentary life, getting exercise by driving instead of walking, had always eaten heartily of rich food, indulged freely in beer and other alcoholic beverages, after the German custom, and had been a heavy smoker. He had never had inflammatory rheumatism or any other illness.

The patient was a man of powerful physique, and in spite of his gray hair did not look at all like an old man. His normal weight was 207, but at date of examination was 190 pounds, while his height was 6 feet. His chest was broad and deep, his bones large and strong, the muscular system well developed, abdomen not corpulent, and subcutaneous fat not excessive. The nails were moderately ridged, the radial arteries stiff but not beady, the temporal and carotid arteries not stiffened, and the subclavians did not stand out prominently nor throb strongly, as they often do in old men. There was a pronounced, visible, and palpable epigastric pulsation reaching at least 2 inches below the xyphoid cartilage, but the apex-beat could not be made out. In the aortic area was a systolic thrill, palpable upon moderate pressure during expiration. The thoracic parietes were so hard and resisting that percussion was difficult, but the lungs were everywhere resonant and respiratory sounds were faint and vesicular. Absolute cardiac dulness was not increased, but by resort to palpatory, auscultatory, and ordinary plessimetric percussion, relative dulness was found greatly increased upward, to the left, and downward, but not notably to the right. The left border reached an inch outside of the left nipple, in all 5 inches from the left edge of the sternum (Fig. 33). With exception of the pulmonic second sound, itself feeble, the heart-tones could not be heard. There was, however, a loud systolic murmur of distinct sawing quality audible throughout the præcordia and for a distance beyond the left nipple into the axillary region. Upon careful study of this murmur it was found to have two areas of maximum intensity, one in the second right interspace near the sternum, the other in the vicinity of the left nipple. Moreover, in these two areas the pitch was slightly yet distinctly different, being lower and harsher in the aortic and more musical in the mitral area. The heart's rate was 90, and its rhythm regular. The liver was palpable.

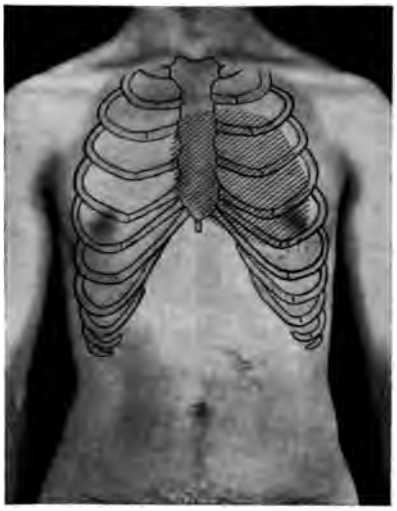


FIG. 33.—RELATIVE DULNESS, CASE OF CHRONIC ENDOCARDITIS (p. 201).

The audible pulmonary second tone and hypertrophic dilatation of the right ventricle confirmed the evidence obtained from the mitral murmur and established the existence of mitral regurgitation. The aortic systolic bruit and loss of the aortic second sound, together with the systolic thrill, gave evidence of stiffness, and perhaps stenosis of the aortic valves. The absence of a rheumatic history, the patient's age, the late development of symptoms, the moderate arteriosclerosis, and lastly, the heart findings, all seemed to warrant the opinion that the valvular changes were due to sclerotic endocarditis. The condition of the kidneys was not ascertained at that time, as the son had not examined the urine, but inasmuch as there was nocturnal micturition, renal cirrhosis was thought probable, and it was advised to have the urine collected for twenty-four hours and examined.

In this case I believe the cause lay in the strain to which the valves of the left heart had been subjected for many years in consequence of the abnormal blood-pressure brought about by his excessive consumption of food and alcoholic liquids without sufficient physical exercise. How much, if any, influence can be attributed to tobacco and waste products I cannot say.

The influence of *strain* has long been recognised in the production of the sclerotic changes now being considered. High blood-pressure, lasting for years, is a cause of valvular as well as of vascular strain, but inasmuch as the individuals in whom such injurious blood-pressure is observed generally lead inactive lives, dine well, and often suffer from indigestion and constipation, it is likely that the products of defective metabolism circulating in the blood act as chemical irritants, and play a not unimportant part in the development of sclerotic changes.

Disease of the aortic valves is frequently observed in men who pursue laborious occupations, as smiths, carpenters, etc., and hence arduous physical exertion is also accredited with the production of valvular and vascular strain and consequent sclerosis. It is in this class of workers that rupture of an aortic cusp is most frequently observed, with its disastrous sequels. It has always seemed to me not an easy thing to correctly estimate the influence of physical strain in working people, since they are so often given to the immoderate use of alcohol and tobacco, and frequently become victims of syphilis. We should probably consider that in these people all these factors are at work, and attribute their chronic endocarditis to their mode of life in general, without attempting to isolate any one etiological factor. Syphilis is undoubtedly capable of setting up sclerotic deformity of the valves, although endocardial changes follow luetic disease far less often than do myocardial and vascular degeneration.

Of that form of chronic endocarditis which is met with in the young, and which is of true inflammatory origin, the one great cause is rheumatism. Although these valvular lesions may undoubtedly begin in an acute vegetative endocarditis, which merges gradually into a chronic process, it is often a low grade of subacute inflammation from the beginning that brings about this form of chronic endocarditis. This inflammation may originate in an acute rheumatic attack, and be recognised clinically at the time, or it may develop so slowly and insidiously as to create no symptoms, and remain undetected for years. Indeed, it is not at all uncommon for valvular diseases originating in this manner to be first diagnosed after compensation has begun to wane. This slowly forming endocarditis gives rise chiefly to stenosis, and, in accordance with the law of numerical frequency, to stenosis of the left auriculo-ventricular orifice.

Physicians sometimes fall into the loose manner of speech of the laity and call the pains of myalgia and an intractable or oft-recurring neuralgia, rheumatic. They should remember, however, that these so-called rheumatic pains are etiologically and pathologically very different from the articular rheumatism that sets up endocarditis. When a student in Munich, I questioned Prof. Joseph Bauer on this subject, and received the emphatic reply that " muscular rheumatism never produces valvular disease." For further discussion of the etiology of endocarditis the reader is referred to the chapter upon Acute Endocarditis and those dealing with the individual valve-lesions.

Symptoms.—The reader of the following chapters will doubtless be impressed by the fact that the different forms of valvular disease present considerable similarity as regards those derangements of circulation of a mechanical nature and those disturbances of visceral function which give rise to subjective symptoms. Such differences as exist are not so much differences in kind as in degree. Any one of the valvular defects may, so long as it is perfectly compensated, exist for years without revealing its existence to the consciousness of the patient, but when compensatory hypertrophy is no longer adequate, conditions result which must of a necessity force themselves upon the notice of the patient with greater or less prominence.

In mitral disease the sensations are mainly due to passive congestion, while in lesions at the aortic orifice they are the result of a diminished or defectively sustained supply of arterial blood; yet in both it would be inaccurate to draw such a strict division. In mitral disease there is defective arterial circulation as well as venous stasis, and when in aortic valve defects compensation fails, there is more or less passive engorgement added to the imperfect arterial flow. Consequently, the clinical picture takes its colouring from both conditions, but in varying proportions, and hence in all forms of valvular disease there comes a time in the stage of destroyed compensation when whatever individual features each affection may have once possessed become blended into the symptom-complex of cardiac inadequacy in its broad sense. Some of these characteristics are plainly recognised as the effects of mechanical pressure in the venous system, as the dull, tense pain of hepatic congestion, the scanty albuminous urine, the hæmorrhoidal congestion and fluxes, and, in large part at least, the serous transudations and the digestive disorders.

Other symptoms are probably owing to the incomplete elimination of the normal products of metabolism; or to the manufacture and accumulation in the system of abnormal products which result from perverted function on the part of the stomach, liver, and other chylopoietic viscera; or there is a combination of these various toxins, with a lessened supply of oxygen and other necessary nutritive principles, that may explain some of the subjective phenomena, such as the dull, oppressive headache, the insomnia, nervousness, and in some instances the low, muttering, or even active delirium occasionally observed in the terminal stage.

The dyspnœa of advanced heart-disease is probably a manifestation of both mechanical pressure in the pulmonary vessels and upon the lungs by the dilated heart and by hydrothorax, but also of deficient oxygenation through sluggish blood-flow and from bronchial obstruction by mucus and serum. Since, then, so many factors enter into the production of the manifold symptoms complained of or manifested by sufferers from valvular heart-disease, it is not possible to satisfactorily account for them all or to explain why some are present in one and absent in another case.

We have also to reckon with individual tendencies, neuroses, intercurrent affections, complications, etc., all of which serve to modify the legitimate clinical picture. For example, I recall a certain young woman who first came under my care for an uncompensated mitral regurgitation of rheumatic origin in 1893, and who during the ensuing five years presented some highly interesting and puzzling phenomena.

At the beginning hers was an ordinary case of mitral insufficiency with slight œdema, which readily yielded to treatment, and she was lost sight of for two years. She then reappeared, having shortly before had a recurrence of rheumatism, and had thereafter been married, both of which occurrences were unfortunate for her. Compensation was so defective, probably in consequence of a fresh endocarditis which had passed beyond its acute stage, and which could be recognised as having existed only by its effects, or in consequence of a pericarditis that had led to adhesion between the sac and anterior chest-wall, that the patient manifested both ascites and anasarca.

Besides this very obvious disturbance of circulation she suffered greatly from insomnia and a degree of emotional instability that could reasonably be considered hysterical and made her extremely hard to control. But the particular feature that puzzled me for a time was the fact that the secretion of urine, scanty at all times, became almost suppressed whenever for the sake of sparing the overburdened heart she was subjected to rest in bed. Whether or not this was due to the abolition of those accessory aids to venous flow residing in muscular movements of the lower extremities and in deepened inspiration incident to gentle exercise about her apartment I could not decide, but this seemed probable from the subsequent fact that the enormous hepatic engorgement and ascites did not disappear until she was given a course of resistance exercises.

Pari passu with this removal of the mechanical hindrance to circulation the insomnia vanished and her normal mental state returned. Compensation was at length regained and retained for a number of months. Six months later, however, she suddenly developed an excruciating and obstinate neuralgia in the course of the right brachial plexus, for which I could discover no adequate cause, and which resisted all treatment. It was accompanied by cough with scanty mucous expectoration, of which repeated careful examinations of the heart and lungs failed to detect any cause aside from the old-standing valvular lesion. At length, discouraged by her failure to obtain relief, she returned to her home in the country, where during the next few weeks she expectorated masses of tenacious sputum, which were said when put in water to spread out and look like the branches of a tree. Whether this was an instance of fibrinous bronchitis or not I cannot say, but certain it is, that when finally her bronchitis subsided her neuralgia also disappeared. I have always believed this was a manifestation of infection, since, as we know, cardiac patients are particularly prone to obscure infections, and that the neuralgia could not be regarded as anywise a symptom directly attributable to her heart-disease.

The next event in this patient's series of experiences occurred cient physical exercise. How much, if any, influence can be attributed to tobacco and waste products I cannot say.

The influence of *strain* has long been recognised in the production of the sclerotic changes now being considered. High blood-pressure, lasting for years, is a cause of valvular as well as of vascular strain, but inasmuch as the individuals in whom such injurious blood-pressure is observed generally lead inactive lives, dine well, and often suffer from indigestion and constipation, it is likely that the products of defective metabolism circulating in the blood act as chemical irritants, and play a not unimportant part in the development of sclerotic changes.

Disease of the aortic valves is frequently observed in men who pursue laborious occupations, as smiths, carpenters, etc., and hence arduous physical exertion is also accredited with the production of valvular and vascular strain and consequent sclerosis. It is in this class of workers that rupture of an aortic cusp is most frequently observed, with its disastrous sequels. It has always seemed to me not an easy thing to correctly estimate the influence of physical strain in working people, since they are so often given to the immoderate use of alcohol and tobacco, and frequently become victims of syphilis. We should probably consider that in these people all these factors are at work, and attribute their chronic endocarditis to their mode of life in general, without attempting to isolate any one etiological factor. Syphilis is undoubtedly capable of setting up sclerotic deformity of the valves, although endocardial changes follow luetic disease far less often than do myocardial and vascular degeneration.

Of that form of chronic endocarditis which is met with in the young, and which is of true inflammatory origin, the one great cause is rheumatism. Although these valvular lesions may undoubtedly begin in an acute vegetative endocarditis, which merges gradually into a chronic process, it is often a low grade of subacute inflammation from the beginning that brings about this form of chronic endocarditis. This inflammation may originate in an acute rheumatic attack, and be recognised clinically at the time, or it may develop so slowly and insidiously as to create no symptoms, and remain undetected for years. Indeed, it is not at all uncommon for valvular diseases originating in this manner to be first diagnosed after compensation has begun to wane. This slowly forming endocarditis gives rise chiefly to stenosis, and, in accordance with the law of numerical frequency, to stenosis of the left auriculo-ventricular orifice.

Physicians sometimes fall into the loose manner of speech of the laity and call the pains of myalgia and an intractable or oft-recurring neuralgia, rheumatic. They should remember, however, that these so-called rheumatic pains are etiologically and pathologically very different from the articular rheumatism that sets up endocarditis. When a student in Munich, I questioned Prof. Joseph Bauer on this subject, and received the emphatic reply that "muscular rheumatism never produces valvular disease." For further discussion of the etiology of endocarditis the reader is referred to the chapter upon Acute Endocarditis and those dealing with the individual valve-lesions.

Symptoms.—The reader of the following chapters will doubtless be impressed by the fact that the different forms of valvular disease present considerable similarity as regards those derangements of circulation of a mechanical nature and those disturbances of visceral function which give rise to subjective symptoms. Such differences as exist are not so much differences in kind as in degree. Any one of the valvular defects may, so long as it is perfectly compensated, exist for years without revealing its existence to the consciousness of the patient, but when compensatory hypertrophy is no longer adequate, conditions result which must of a necessity force themselves upon the notice of the patient with greater or less prominence.

In mitral disease the sensations are mainly due to passive congestion, while in lesions at the aortic orifice they are the result of a diminished or defectively sustained supply of arterial blood; yet in both it would be inaccurate to draw such a strict division. In mitral disease there is defective arterial circulation as well as venous stasis, and when in aortic valve defects compensation fails, there is more or less passive engorgement added to the imperfect arterial flow. Consequently, the clinical picture takes its colouring from both conditions, but in varying proportions, and hence in all forms of valvular disease there comes a time in the stage of destroyed compensation when whatever individual features each affection may have once possessed become blended into the symptom-complex of cardiac inadequacy in its broad sense. Some of these characteristics are plainly recognised as the effects of mechanical pressure in the venous system, as the dull, tense pain of hepatic congestion, the scanty albuminous urine, the hæmorrhoidal congestion and fluxes, and, in large part at least, the serous transudations and the digestive disorders.

Other symptoms are probably owing to the incomplete elimination of the normal products of metabolism; or to the manufacture and accumulation in the system of abnormal products which result from perverted function on the part of the stomach, liver, and other chylopoietic viscera; or there is a combination of these various toxins, with a lessened supply of oxygen and other necessary nutritive principles, that may explain some of the subjective phenomena, such as the dull, oppressive headache, the insomnia, nervousness, and in some instances the low, muttering, or even active delirium occasionally observed in the terminal stage.

The dyspnœa of advanced heart-disease is probably a manifestation of both mechanical pressure in the pulmonary vessels and upon the lungs by the dilated heart and by hydrothorax, but also of deficient oxygenation through sluggish blood-flow and from bronchial obstruction by mucus and serum. Since, then, so many factors enter into the production of the manifold symptoms complained of or manifested by sufferers from valvular heart-disease, it is not possible to satisfactorily account for them all or to explain why some are present in one and absent in another case.

We have also to reckon with individual tendencies, neuroses, intercurrent affections, complications, etc., all of which serve to modify the legitimate clinical picture. For example, I recall a certain young woman who first came under my care for an uncompensated mitral regurgitation of rheumatic origin in 1893, and who during the ensuing five years presented some highly interesting and puzzling phenomena.

At the beginning hers was an ordinary case of mitral insufficiency with slight œdema, which readily yielded to treatment, and she was lost sight of for two years. She then reappeared, having shortly before had a recurrence of rheumatism, and had thereafter been married, both of which occurrences were unfortunate for her. Compensation was so defective, probably in consequence of a fresh endocarditis which had passed beyond its acute stage, and which could be recognised as having existed only by its effects, or in consequence of a pericarditis that had led to adhesion between the sac and anterior chest-wall, that the patient manifested both ascites and anasarca.

Besides this very obvious disturbance of circulation she suffered greatly from insomnia and a degree of emotional instability that could reasonably be considered hysterical and made her extremely hard to control. But the particular feature that puzzled me for a time was the fact that the secretion of urine, scanty at all times, became almost suppressed whenever for the sake of sparing the overburdened heart she was subjected to rest in bed. Whether or not this was due to the abolition of those accessory aids to venous flow residing in muscular movements of the lower extremities and in deepened inspiration incident to gentle exercise about her apartment I could not decide, but this seemed probable from the subsequent fact that the enormous hepatic engorgement and ascites did not disappear until she was given a course of resistance exercises.

Pari passu with this removal of the mechanical hindrance to circulation the insomnia vanished and her normal mental state returned. Compensation was at length regained and retained for a number of months. Six months later, however, she suddenly developed an excruciating and obstinate neuralgia in the course of the right brachial plexus, for which I could discover no adequate cause, and which resisted all treatment. It was accompanied by cough with scanty mucous expectoration, of which repeated careful examinations of the heart and lungs failed to detect any cause aside from the old-standing valvular lesion. At length, discouraged by her failure to obtain relief, she returned to her home in the country, where during the next few weeks she expectorated masses of tenacious sputum, which were said when put in water to spread out and look like the branches of a tree. Whether this was an instance of fibrinous bronchitis or not I cannot say, but certain it is, that when finally her bronchitis subsided her neuralgia also disappeared. I have always believed this was a manifestation of infection, since, as we know, cardiac patients are particularly prone to obscure infections, and that the neuralgia could not be regarded as anywise a symptom directly attributable to her heart-disease.

The next event in this patient's series of experiences occurred about a year later. She had again been suffering from rheumatism, as I was told, when, according to her sister's statement, she suffered one night from severe pain in the region of the heart, for which hot cloths were being applied. Suddenly the sufferer exclaimed, "There! something has broken, and the pain has all gone." She then seemed to sink away, her pulse becoming too weak and rapid to be counted, her extremities cold, and her countenance blue. Stimulants revived her, but all night she continued to have sinking spells, which necessitated the administration of restoratives.

The explanation of this attack has never been clear to me. Whether a tendinous cord snapped, or a pericardial adhesion gave way, or whether the pain may not have been due to a muscular cramp, I cannot say. Under the influence of heat the muscle may have suddenly relaxed, and thus caused a sensation, which to the suffering and highly nervous apprehensive girl was naturally attributed to the heart, and threw her into a condition of mental shock which reacted on the weakened heart. At all events, the attack was fraught with no less alarm to the friends than to the patient, and an explanation was sought, which could not be given.

Six weeks subsequently the patient was again brought to the city in a truly deplorable condition. There were marked evidences of cardiac asthenia and consequent circulatory embarrassment, pronounced icterus, œdema of the ankles, ascites, enormous hepatic congestion, œdema of the left but not the right arm, and in the heart signs of double mitral disease, relative tricuspid insufficiency, and adherent pericardium.

The feature of chief interest, however, was connected with the dropsy of the left upper extremity. This was strictly localized, extending from the fingers up to and ceasing with the shoulder. Palpation of the axilla disclosed that the axillary vein had been converted by thrombosis into a firm cord. To what distance the thrombosis extended down the arm could not be determined, but it did not involve the jugular veins. The "swelling," it was stated, had made its appearance a week earlier, but aside from the annoyance did not appear to occasion pain or distress.

This highly interesting and comparatively rare condition was an instance of venous thrombosis occurring in some cases of valvular disease. It has formed the subject of an instructive paper by Dr. William Welch, which was read at the session of the Association of American Physicians in 1900. Welch was able to collect but 28 recorded instances, including his own, although, as stated by him, the condition probably occurs more often than it is recognised. Of these 28 cases, all but 4 involved the veins of the upper extremities and neck, a fact which lends to it additional interest and importance. Twenty-two cases showed thrombosis of the left side alone 15 times, bilaterally 8 times, while only twice was it confined to the right. Welch found that the thrombosis might be limited to the veins of the arm, to those of the neck, or might involve all the veins—that is, the superior vena cava, the innominate, both internal and external jugular, subclavian, axillary and brachial, and even, as in one case, the superior thyroid.

Although thrombosis may and does sometimes occur in individuals suffering from chronic arteriosclerosis and nephritis, yet in Welch's 28 cases there was in every instance valvular disease as follows: Mitral regurgitation 9 times, mitral stenosis alone 6 times, mitral stenosis with insufficiency 6 times, and aortic regurgitation with relative mitral incompetence once. In 10 instances there was associated aortic and mitral disease. The thrombus was either red or reddish-gray, and although in some instances it was softened at its centre, it for the most part was firm throughout, and occluded the vessel excepting at its extremities. In one case it was a "wall thrombus." It is also interesting to note that the thrombosis appeared to have begun at the lower end of the internal jugular in those instances in which it involved the cervical veins. This fact led Welch to conclude that the formation of the thrombus was favoured by the peculiar anatomical arrangement of the cervical veins on the left side, together with the conditions governing the blood-flow in them.

As pointed out by Hanot, the left innominate vein is longer and more oblique than the right, which, together with the right-angle junction of the left internal jugular with the subclavian and the bulbous expansion of the internal jugular, favours, in Welch's opinion, the formation of eddies or whirling currents at that point, and thus furthers the development of thrombosis. Moreover, he thinks there is probable pressure upon the subclavian vein by the dilated left auricle and dilated pulmonary veins, so that circulation in the cervical and arm veins becomes extremely sluggish, and thus provides another favouring factor. Finally, in one of his cases Welch was able by cultures to identify the streptococcus pyogenes, which, he thinks, warrants the hypothesis that in these cases there is an infectious origin for the thrombosis, a conclusion which is strengthened by recent observations going to show that in all cases of venous thrombosis there is an infection.

In my case there is good reason to believe that the patient was still suffering from some infection, for she had but a few weeks earlier gone through with what was called rheumatism by her home physician, yet which may very well have been a streptococcus infection, which so often presents the appearances of articular rheumatism. Moreover, there were three small, distinctly indurated lymphatic glands situated just above the left clavicle, near the outer border of the left sterno-cleido-mastoid muscle, while the patient displayed a slight elevation of temperature. In other respects my case conformed with the most of Welch's requirements —namely, she was a female, of but twenty-three years of age, and was a sufferer from mitral disease. Her symptoms too were characteristic in the localization of the œdema to the affected arm below the location of the thrombosis. She did not, however, suffer pain, at least not at the time the interesting condition was detected, the occluded vein was not tender, and I failed to discover any enlargement and tenderness of the lymphatics of the arm, as is often present. If such existed, they were hidden from observation by the œdematous condition of the extremity. Welch states, finally, that in at least one of the cases collected by him there was mild delirium, which was attributed to the cerebral œdema discovered at the autopsy.

In the matter of the diagnosis of this form of venous thrombosis there is no difficulty, provided the thrombosed vein can be felt, and even when not, strictly localized dropsy in one arm or one side of the neck renders the existence of thrombosis very likely. Nevertheless, according to Hanot, the greater length and obliquity of the left innominate vein may sometimes cause unilateral and circumscribed œdema even when venous thrombosis is not present.

The prognosis is unfavourable to recovery from the dropsy if the veins are extensively plugged. If the thrombosis is not complete, or is of limited extent, it is possible for collateral circulation to become established and absorption to take place. Finally, the condition is likely to occur in the terminal stage of the valvular disease, and if very extensive it may contribute to the patient's death.

It is not common for patients with chronic valvular disease to suffer from embolism, and yet such a possibility should always be borne in mind. It is stated that such an occurrence is more frequent in mitral than aortic disease; and I have under observation a female patient with mitral insufficiency who has permanent contracture of the fingers of the left hand and but partial use of the arm as a result of an embolus that was thrown off presumably from her mitral valve nearly six years ago. The symptoms of embolism are usually said to be pain in the part where the plug lodges, nausea, and even vomiting, a chill, and rise of temperature. To judge from cases of embolism observed in acute endocarditis and from pulmonary infarcts, I should say that pain in the affected part is the most constant symptom.

The splenic artery is a frequent seat of embolism, and it may well be that the transient pain from which cardiac patients not infrequently complain in the region of the spleen may be of this origin. It is unsafe to make such a diagnosis, however, unless one can detect enlargement and tenderness of this organ following the pain. This is emphasized by the fact that these patients are very prone to sudden and sharp pains of a neuralgic character in various situations, particularly in the abdomen.

I recall an instance that was narrated to me by an ophthalmologist of sudden blindness of one eye resulting from the plugging of the retinal artery, and as the lady possessed a blowing systolic apex-murmur, the embolus was thought to have been a minute vegetation from her mitral valves. I have also been informed of a young lady with valvular disease who, upon awakening one morning, was found to have lost during the night all recollection of certain members of her own family. This peculiar lapse of memory was attributed by her medical attendant to embolism.

Pulmonary infarcts are not at all uncommon in cases of advanced valvular disease, and are evinced by sudden acute pain in the affected lung, together with frequent cough and the spitting of clear blood. These cases must not be confounded with instances of hæmoptysis due to sudden increase of pulmonary congestion, as not seldom occurs in mitral patients who have overtaxed their hearts. In these cases the history of some exertion and the absence of sudden, sharp pain will usually aid in the differential diagnosis. Embolism of the middle cerebral artery is attended by such manifest symptoms that there is usually but little difficulty in determining the cause of the phenomena.

In a single instance I have observed the conjunction of true epilepsy with valvular disease. The patient was a man of about forty who presented well-marked signs of mitral stenosis. His valvular lesion was of rheumatic origin, and his convulsions antedated his cardiac disease by some years. There was every reason to conclude that the association of these two affections was purely accidental as regards their etiology. The fits were usually excited by indiscretions in diet, and required bromides for their control. Although never assuming any causative relation between the two, I yet believed they exerted a deleterious influence upon each other. I am very certain that the epilepsy affected his mitral disease unfavourably by serving to maintain and aggravate the dilatation of the cardiac chambers. Such cases as this are not to be regarded as instances of cardiac epilepsy, which term has been employed to designate attacks of præcordial pain accompanied by loss of consciousness and succeeded by twitchings of the muscles of the face.

The association of epilepsy and heart-disease in my case serves to emphasize the fact that valvular diseases may be complicated and have their clinical picture modified by other affections. I speak of this because inexperienced physicians are apt, when treating patients with valvular disease, to attribute all symptoms to the cardiac complaint.

Cardiopaths frequently become anæmic and neurotic, hysterical or neurasthenic, and then complain of all sorts of sensations, which it is clearly impossible to attribute to their valvular affection.

The French describe what they term "cardiac cachexia" in distinction from cardiac asthenia, and which is analogous to the cachexias of malignant or tuberculous disease. They ascribe it to some chemical change in the blood. It is not at all uncommon to see cardiac patients who, while presenting no very marked symptoms of cardiac inadequacy, yet display an appearance of malnutrition that might not inaptly be termed a cachexia. In such there are often symptoms of weakness, inability to take and assimilate food, and various other features that, even if referable primarily to a valvular defect, are nevertheless attributable to their general state rather than to their heart.

Patients with valvular disease sometimes become victims of attacks of palpitation and præcordial pain that place them in the category of cardiac neuroses, although, strictly speaking, this term should be applied to cases in which the attacks are independent of any discoverable heart-disease. At such times there may even be irregularity or intermittence of the pulse. This is naturally thought due to the cardiac lesion, and yet it may be wholly independent of the heart disorder, being the result of some toxin perhaps, or of some obscure nervous excitation. This is proved by the observation that, when the attack subsides, the heart's action returns to its former regular and tranquil state. I recall such an instance in a young man with a mitral regurgitation who was subject to attacks of palpitation that invariably threw him into a perfect panic of fright and apprehension. Yet when his attacks subsided he resumed his ordinary duties without a symptom of his cardiac lesion.

I am in the habit of assuring such patients that their attacks of palpitation are not indicative necessarily of a serious state of the heart—for persons without any demonstrable cardiac disease often manifest similar disturbance—but that because the heart is not structurally sound, it is more easily thrown out of balance by indigestion or other conditions that would not be noticed were it in perfect health. Nothing is worse for cardiopaths than to get into a state of introspection and constant apprehension. I have known some who were so alarmed over their valvular defect that they might be said to be possessed by a veritable phobia. Such patients not only imagine all sorts of symptoms that play no part in the clinical history of their particular lesion, but they are afraid to venture out alone lest they get an attack or be brought home dead. The medical adviser should therefore carefully distinguish symptoms due to the valve-defects from those that belong to some associated disorder, and reassure the patient accordingly.

Before completing this subject I desire to add a few remarks concerning derangements in the rhythm of the heart's action.

Irregularity and intermittence of the pulse are common in all forms of valvular disease, yet they are by no means always observed. The regular and rhythmic contraction of the heart-muscle depends upon its receiving regular and uniform stimulation by the presence of the blood. Doubtless there are many disturbing factors of which we are yet ignorant, and which may be transient in action, such as emotional or other impulses acting through the nervous system, toxins of various kinds, etc. But aside from these there are degenerative or other alterations of the cardiac muscle itself that lead to arrhythmia. Such a persistent derangement of the pulse is therefore an unfavourable omen. It is quite likely to be observed in mitral disease, particularly regurgitation, it is said. Why this is, is not clear, unless it is dependent upon dilatation or degeneration of the auricles.

A mere lack of uniformity in the force, volume, and frequency of the pulse-waves, to which form of arrhythmia is applied the term irregularity, is far less serious than intermittence, by which is meant a dropping out of some of the pulse-waves. This latter may be due to actual intermissions in the cardiac contractions, or it may be caused by the failure of all the blood-waves to reach the wrist, the heart itself beating regularly all the time, although with unequal force. Many interesting varieties of pulse-rhythm have been described, but I do not know that they possess any special clinical significance aside from the fact of their being an indication of disordered cardiac action. Thus *pulsus alternans* is a term employed to describe an irregularity consisting in the appearance first of a large and then of a small wave that follow each other in regular succession. When the pulse-waves occur in pairs that are separated by distinct intervals they are spoken of as *pulsus bigeminus*, and when in groups of three as *pulsus trigeminus*. *Pulsus intercidens* or *intercurrens* denotes the occurrence or interpolation of occasional small waves between the regularly occurring large waves. *Pulsus tardus* is a slow pulse, *magnus* a large pulse, *parvus* a small pulse, etc.

These variations may exist alone and for a considerable time, or they may be blended and display their individual characters for but a few seconds; so that a pulse results that is strikingly irregular and difficult or impossible to count.

It has been my observation that when a patient with organic heart-disease displays a persistently arrhythmic pulse he is usually not aware of its existence by any sensations within his chest. On the contrary, persons with so-called functional derangement are very apt to experience a sensation as if the heart jumped or turned over whenever it intermits.

In studying the pulse in any case of valvular disease it is not only necessary to observe its rhythm, but one should take particular notice of its force and volume. In conditions of stenosis the pulse is very likely to be small and of poorly sustained tension, showing that the arterial system is defectively flushed. In some cases, however, of mitral stenosis the pulse is small and tense in consequence of obstruction to the flow of blood out of the capillaries. Peculiarities of the pulse of aortic regurgitation will be dwelt upon at some length in that section.

CHAPTER VI

MITRAL REGURGITATION

By this term is designated an abnormal escape or leakage of blood from the cavity of the left ventricle through the left auriculo-ventricular orifice into the left auricle. Such regurgitation may be due to structural defect of the valves or to stretching of the ostium, in consequence of which the valve-segments are rendered relatively incapable of adequately closing the opening. In this chapter will be considered only the form due to organic defect. Other terms applied to this disease are mitral incompetence or insufficiency; but inasmuch as the term regurgitation is more commonly employed in this country, this is the one that has been selected and is preferred.

Morbid Anatomy.—The structural changes of the mitral valve permitting regurgitation of the blood are mainly thickening of the cusps with increased rigidity that prevents perfect coaptation, or the shortening or retraction of one or both of the leaflets in such a way as to permit the reflux of blood. The segments lose their normal pinkish transparent appearance, thin delicate feel, and become thickened, stiff, and of an opaque whitish or grayish colour. Contraction of the fibrous tissue may cause retraction or shortening of one or both of the cusps, or curling of their edges in a manner to prevent effective closure of the valve. In the valvular disease following acute endocarditis the shrivelled and often calcified remains of old vegetations may be found on the auricular surface of the valve along the line of maximum contact. When these old vegetations are numerous the mitral ring is usually fibrous and contracted, leading to a condition of stenosis as well as regurgitation (Fig. 34). Very commonly, also, the tendinous cords are found more or less matted together, stiffened, and shortened, so as to still further interfere with perfect action of the valve. The deposit of calcareous matter is by no means limited to the old vegetations, but may affect the valve-cusps, the chordæ tendineæ, or even the mural endocardium in the neighbourhood. In fact, the deposit of lime-salts may in some instances be so extensive as to convert the entire valvular apparatus into a firm calcareous mass, having no resemblance whatever to the original structure.

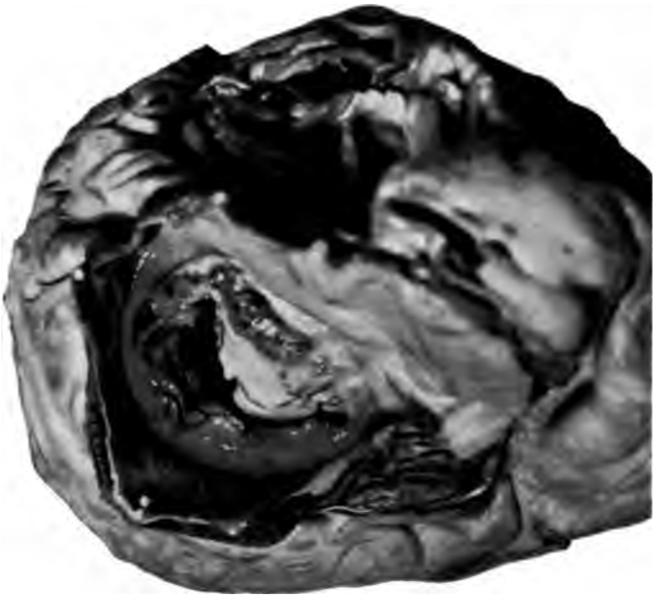


FIG. 34.—SHOWS CONDITION OF MITRAL VALVE, CAUSING REGURGITATION AND OBSTRUCTION. LEFT AURICLE HAS BEEN DISSECTED AWAY.

The local changes in the endocardium, however, form but a small part of the morbid changes found in a case of mitral insufficiency. The changes in the circulation, and the effect on the heart-wall and on the other organs of the body that were considered in a general way under chronic endocarditis, take place here and require description. Whenever mitral regurgitation occurs a portion of the contents of the left ventricle is forced back during systole into the left auricle, which at the same time is receiving the normal flow of blood from the pulmonic veins. The chamber is consequently surcharged, and as the two streams enter it during its diastole it becomes overdistended. At the same time, in consequence of its dilatation, the auricle has greater work to perform in emptying itself of this increased amount of blood, and in accordance with the physiological law that an organ which has increased work to do will, so long as its nutrition is unimpaired, manifest increased power for work, the walls of the auricle at length become thicker and stronger. In time, therefore, the auricle comes to be both hypertrophied and dilated.

If these changes in the circulation come on slowly, and time is afforded for compensatory changes in the heart to develop, the nutrition of the heart-muscle, and indeed the integrity of the circulation, may suffer no serious injury. Furthermore, if the auricle is able to deliver an increased volume of blood to the ventricle, this chamber is able to discharge into the aorta, in spite of the regurgitation, an approximately normal amount of blood, and adequate arterial circulation is maintained. So far, then, the hypertrophy and dilatation of the auricle compensates the valvular defect, which may consequently exist for a long time without producing any inconvenience.

If, however, the leakage is extreme, or if it becomes so with lapse of time and the auricle is unable to completely empty itself, a residue remains, which interferes with the inflow of blood from the pulmonary veins. Thus takes place an accumulation of blood (passive congestion) which, acting as increased peripheral resistance to the work of the right ventricle, leads to hypertrophy and dilatation of this chamber. Moreover, the stasis within the lungs induces in them the condition known as brown induration, in which the connective-tissue elements are increased, the veins engorged, and the whole organ is of a dark-brown colour in consequence of pigment deposited by the disintegrating blood. In advanced stages there may also be pulmonary œdema and hydrothorax from transudation of serum out of the engorged vessels. There is always more or less bronchial congestion in consequence of stasis within the pulmonic vessels.

When at length blood-pressure in the pulmonary artery grows excessive the wall of the right ventricle finds its work too great, and, yielding to the strain, permits dilatation to supersede hypertrophy. This chamber is now unable to fully empty its contents, stasis within it grows, and at length causes such dilatation that the tricuspid orifice stretches, and its valve is no longer able to close adequately. In cases of long standing and extreme pressure in the pulmonary artery, stretching of its ostium is also occasioned, and leads to relative pulmonary incompetence.

So long as the left auricle and right ventricle are capable of coping successfully with the back wash from the left ventricle the work of the right auricle is not especially increased. When, on the contrary, the right ventricle begins to yield to the strain, particularly when it becomes dilated, back pressure is exerted upon the right auricle and great venous system. Veins everywhere grow more or less turgescent, and the internal organs display the effects of engorgement. The liver in particular becomes enlarged and in time indurated, and on section shows the peculiar mottling that has given to the organ in this condition the name of nutmeg-liver.

In the stomach and intestines passive congestion leads to chronic catarrh of the mucosa, and in the kidneys to cyanotic induration or even chronic nephritis. None of the internal viscera escape, while the veins share in the distending effects of stasis and become relatively larger than the arteries. Back pressure creeps downward into the vessels of the lower extremities, and as circulation grows still more sluggish serous transudation finally makes its appearance. Commencing in the feet, dropsy gradually extends upward, invades the peritoneal cavity and walls of its contained viscera, and in extreme cases the serous cavities within the chest, and finally the lungs (Fig. 35). Thus far we have considered the secondary effects that are produced behind the seat of the original lesion. There are, in addition, certain effects of mitral regurgitation in front of the lesion. These are the hypertrophy and dilatation of the left ventricle found in cases of free and uncombined mitral insufficiency. This enlargement seems rather remarkable at first thought, since one would naturally think the chamber ought to be smaller rather than larger in size. At one time this condition of hypertrophic dilatation was explained on the hypothesis that in consequence of venous and capillary stasis blood-pressure was increased in the arterial system, and that hence there was augmented intraventricular pressure which resulted in dilatation, with increased demand for work which led to hypertrophy.

This theory is now known to be incorrect, and has been replaced by the following: Owing to the abnormal volume of blood contained by the left auricle at the close of its diastole, this chamber, which has become hypertrophied, discharges with great force an unnatural amount of blood into the ventricle. This

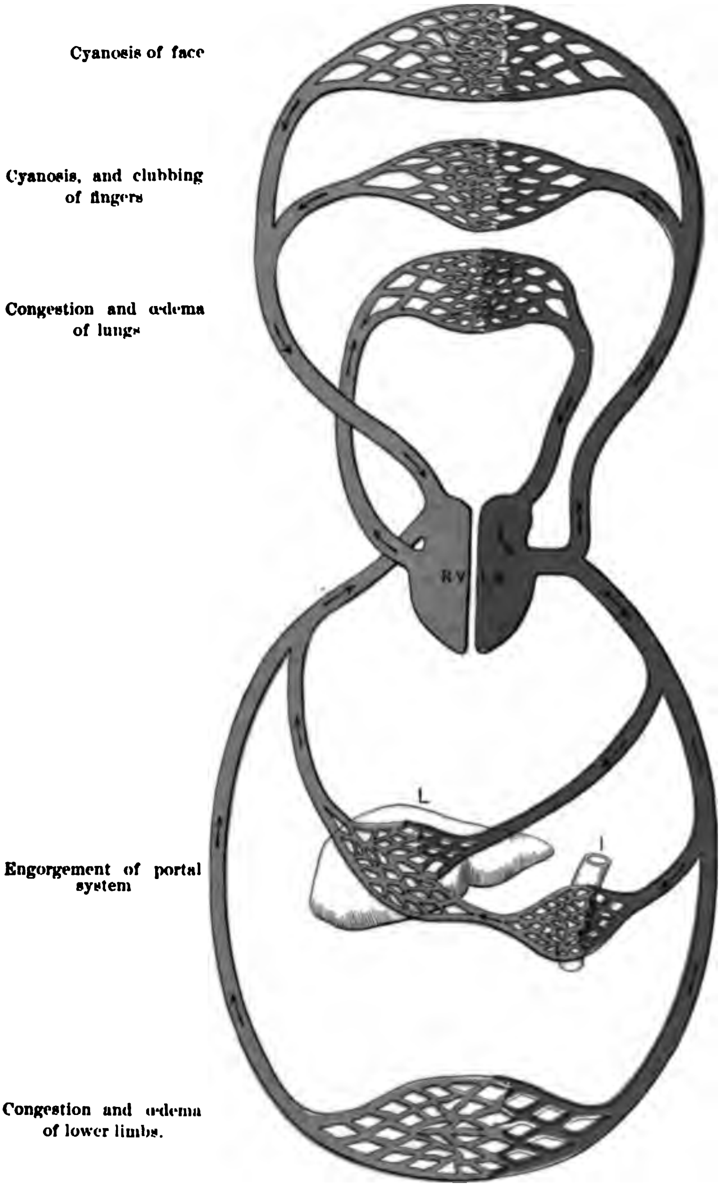

Cyanosis of face
Cyanosis, and clubbing of fingers
Congestion and œdema of lungs
R V
L
I
Engorgement of portal system
Congestion and œdema of lower limbs.

Fig. 35.—Diagram showing Effects on the Circulation of a Mitral Leak.

cavity is in a state of diastole when it receives this inrush, and, being relaxed, becomes after a time dilated. At the same time it is forced to handle a larger volume of blood, which it can only do by undergoing hypertrophy, and thus at last this ventricle comes in its turn to feel the secondary effects of the circulatory disturbance.

In time, moreover, when stasis has become everywhere apparent, the left ventricle undergoes still further dilatation, for which it has become prepared by certain structural changes within its myocardium. Its myocardium is flabby and of a brown instead of the normal beefy red colour, while its fibres are found microscopically to be reduced in size and to contain granules of brown pigment, especially near the nucleus. This increased dilatation of the ventricle at this time is explained by some as due to the high blood-pressure in the arterial system secondary to stasis in the veins, which abnormal arterial blood-pressure interferes with the easy emptying of the ventricle. This is a defective explanation, however, since physiologically the abnormal blood-pressure in the venous system leads to lowered instead of heightened blood-pressure in the arteries. The dilatation of the left ventricle is now due to the weakness of its own wall, which does not permit it to completely empty its cavity with each systole. Thus is established a residue which augments the amount of blood received from the auricle with the next diastole, while at the same time its wall is powerless to withstand the dilating force of this stream that pours into it. Thus is at length set up a vicious circle in consequence of which the effect of the original valvular incompetence intensifies the regurgitation.

The typical heart, then, of mitral insufficiency is enlarged. The enlargement is mostly of the right ventricle, and the organ has in consequence a rounded apex. The tricuspid orifice, and often the pulmonary, is found to be wider than usual, owing to the dilatation of the right ventricle. The left ventricle is moderately and the left auricle greatly enlarged, while the mitral valve shows the structural changes already described, which have been the cause of the whole trouble.

Etiology.—What has already been said concerning the causation of chronic endocarditis applies equally to mitral insufficiency, since this is but one of the manifestations of that affection. I shall therefore only add a few general considerations bearing on the localization of the deforming process at the mitral orifice.

Incompetence of the left auriculo-ventricular valve is a very frequent cardiac affection—is indeed the most frequent of all valvular defects. This is particularly the case in children and young adults, forming in this period of life the counterpart in point of frequency of the sclerotic changes at the aortic orifice in persons, chiefly men, past middle age. The influence of childhood and youth in the generation of mitral regurgitation lies doubtless, not in the fact of the age, *per se,* but in the prevalence in the young of those diseases, inflammatory rheumatism, chorea, and the exanthemata, which set up endocarditis. The greater liability of the mitral than of the aortic valves to suffer from endocardial inflammation in the earlier decades of life is probably owing to their being exposed to relatively greater strain, which their more delicate structure fits them less well to endure.

As regards sex, it is generally stated that mitral regurgitation is more frequent among males than females, but in analyzing my case-records I find no predominance of either sex. After throwing out all cases that from the history, age, or symptoms cannot be safely considered as organic, there remain 126 cases in which regurgitation was due to structural alteration of the valves. These were divided equally between the two sexes. Classifying these 126 cases according to decades, there were 6 boys and 6 girls between one and ten years of age, 12 males and 16 females between ten and twenty, 18 of each sex between twenty and thirty, 14 each between thirty and forty, and 15 males and 7 females over forty. Examined with reference to rheumatism and other diseases, it was found that 32 males and 28 females gave a history of rheumatism alone, 4 males and 6 females of scarlatina alone, 5 males and 4 females of measles alone, 1 female of chorea alone, while of more than one disease 1 male and 3 females had had rheumatism and scarlatina, 4 each had had rheumatism and measles, and 4 each rheumatism, scarlatina, and measles, 2 males and 5 females scarlatina and measles, 2 females measles and pertussis, and 1 female all four diseases, 2 males and 5 females chorea, in combination with some of the other diseases mentioned. Two males gave a history of venereal disease. Of the remaining cases, in which no definite history of previous disease could be elicited, the organic nature of the lesion was rendered probable either by the existence of arteriosclerosis or by the youthful age of the patient and the absence of anæmia or other factors pointing to a relative mitral insufficiency.

Symptoms.—The presence or absence of distinctively cardiac symptoms depends upon the degree of the leak and of the compensatory hypertrophy that has been established. Consequently we have to distinguish cases in which subjective manifestations of circulatory disturbance are wanting from those in which there is more or less evidence of cardiac inadequacy. In the former class such symptoms as are complained of are probably referable indirectly to the valvular defect, but are nevertheless such as we encounter in persons without disease of the heart. In some instances they direct the experienced physician's attention to the possibility of mitral disease, while in others they seem to point rather to disorders of other organs, and the discovery of the regurgitation is accidental. In such cases the individual first learns of his malady on applying for life insurance, or upon subjecting himself to physical examination preparatory to athletic training, or upon consulting his physican for some trifling ailment. The damage to the valve is slight and compensatory hypertrophy is perfect. It would in some instances be better for such persons not to be informed of their defect, since although they are able to endure games of skill and considerable exertion, as tennis, without conscious symptoms, they are likely after learning of their lesion, particularly if of a nervous, excitable temperament, to be alarmed by palpitation, which prior to their knowledge did not attract their attention.

In other instances regurgitation is free, yet there is a truly remarkable absence of subjective consciousness of its existence. This is generally due to the completeness of the compensatory hypertrophy on the part of the right ventricle and the left auricle. Yet I have known individuals of a not very impressionable temperament who, in spite of rather inadequate compensation, were unconscious of symptoms referable directly to their mitral disease. Some excitable neurotic persons, like those first alluded to, consult physicians for symptoms referable to the digestive tract, or nervous system, rather than to the heart itself. These are anorexia, or discomfort after food, constipation, or an irregular state of the bowels, distressing præcordial pains, which either set up or are accompanied by palpitation, and which greatly alarm the patient and friends. In a multitude of such cases there is no objective evidence of loss of compensation, and the patients are able to enjoy outdoor sports or to participate in feats of endurance without subjective symptoms.

Again, cases occur in which the only symptoms are such as are usually classed under the head of lithæmia, or the irregular manifestations of gout, and occurring in persons of sedentary habits, are removed by regular outdoor exercise.

In other cases the most that can be said is they appear to have an unstable nervous system, and their symptoms in nowise differ from those of other individuals of the same category whose hearts are healthy. In all such the valvular lesion does not appear to be directly responsible for the manifestations, and yet it may well be that these can be referred to defective nutrition and elimination in consequence of the circulatory disturbance.

It is not uncommon for females with organic mitral insufficiency to present evidence of simple secondary anæmia or of chlorosis without symptoms of cardiac inadequacy. If in such cases there is shortness of breath upon unusual exertion, it is no greater than may reasonably be attributed to the blood-state. I recall a remarkably interesting and instructive instance of this kind. In March, 1901, a young married lady of twenty-four sought my opinion because of " a grating sound in the heart," which first attracted her notice in the fifth month of her pregnancy, and which still annoyed her at times of unwonted physical effort, as during rapid walking. Aside from this symptom, there was nothing that made her conscious of her heart. She admitted getting a little out of breath, but this was so slight she had not given it any attention. Both her parents had died of pulmonary tuberculosis, but of her brothers and sisters, seven in all, none had shown signs of the disease. She had had pneumonia when but a year old, scarlatina at eight, measles and pertussis in childhood, but never rheumatism, and up to the time of her marriage, at twenty-two years of age, she had considered herself well, being able to romp and play like other children without trouble. With exception of the " grating sound " mentioned, her pregnancy and confinement had been uneventful, and she could not recall having suffered from more dyspnœa than do other women towards the later months of pregnancy. She had nursed her baby for nine months, and during that period lost 29 pounds, of which seven had been regained in the seven months following the weaning of her infant. Her appetite was poor, bowels were irregular, and she was apt to suffer from sour stomach and eructations. The menses were regular but scanty. Hands and feet were generally cold, and she said she had grown nervous, being easily excited. Pain of any kind was trifling, forming a marked contrast to most of the cases I encounter.

In all this history and description of symptoms there was nothing to point to the heart outside of her declaration that she sometimes heard a queer sound, which she wanted to learn the meaning of. The pulse was 85, equal, regular, but too small and weak. The broad, strong apex-beat was in the normal situation, and no increase of either superficial or deep cardiac dulness could be made out. However, the first sound was partly obscured by a loud, rasping murmur that was audible throughout the cardiac area and transmitted around the left side to the lower angle of the scapula. The pulmonary second sound was accentuated, and in the dorsal decubitus a softer blowing systolic murmur could be heard in the pulmonary area. Thinking this last might be a chlorotic murmur, I had the blood examined, and found that the hæmoglobin was reduced to 65 per cent, red and white cells being normal in number.

In spite of the absence of a rheumatic history and despite chlorosis, there seemed no good reason to doubt the existence of a mitral regurgitation of endocarditic origin, but as compensation was preserved, the patient was reassured as to the harmlessness of the sound she had noticed. In fact, she was given to understand that the more serious conditions were the blood-state and loss of weight, which in the light of her family history certainly required attention. She was given a little strychnine and a few drops of digitalis to improve the strength of her pulse, but main attention was bestowed upon the matter of nutrition. Milk, raw eggs, and fresh air were insisted upon. The patient obeyed instructions to the letter, and soon was disposing admirably of two quarts of milk and ten raw eggs daily in addition to three good meals. Her colour, weight, and general condition improved steadily, until at the end of two months she looked the picture of health. Nevertheless, the murmur persisted, and once in a while she heard that same endocardial sound. It no longer worried her, however, and she never complained of any other symptoms referable to the heart or disordered circulation.

In this case it would be difficult to say how much the mitral regurgitation was responsible for her condition. I believe the leak was so slight that it did not materially affect the chylopoietic and blood-making organs, but that the state of her general health was attributable to her child-bearing and lactation, which in a woman with hereditary predisposition to pulmonary tuberculosis proved too great a draught on her vitality. Had she been allowed to go on in her reduced condition she would in time have developed symptoms either of cardiac inadequacy or of tuberculosis. As it is, she is now likely to remain free from symptoms of valvular disease for an indefinite time.

I have notes of the case of a young man who, because of a mitral regurgitant murmur and not very severe symptoms of cardiac strain, was ordered to bed by his physician, and there remained for two years. When I saw him he had mitral insufficiency sure enough, but his prolonged rest had established perfect compensation, the heart being not demonstrably enlarged and the liver of normal size. Yet he declared he could not get up or stand, much less walk. I compelled him to get on to his legs, and little by little to walk about, with the result that he found he could exercise without harm or symptoms of heart-weakness. He was easily frightened about himself for two or three years, but did not manifest dyspnœa or other cardiac symptoms even when riding his wheel over hilly and sandy roads. The last time he was seen by me, now several years ago, he was as well as nine-tenths of the young men who, like him, are school-teachers. At the most he had to be careful of his stomach and guard against constipation.

A medical student, aged thirty-four, sought medical opinion on account of attacks of palpitation. In childhood he had measles, pertussis, and chicken-pox, and dimly recalled having had a swollen knee when five years old. At twelve or thirteen was so puny and frail that he was given cod-liver oil and kept in the house during the winter, but after fourteen took a start and became rugged. Fourteen years ago he had gonorrhœa; had used tobacco from the age of seventeen. In 1893, 1894, and 1895 he played football a good deal, and again two years ago, 1898, without any distress connected with his heart, excepting on one occasion when, after having drunk a number of glasses of beer, he played in a very rough game. He then noticed considerable shortness of breath and several times had to drop out and lie down on the grass, because experiencing great difficulty in getting breath. He attributed his dyspnœa to the fact of having attempted to play on a full stomach. About a month later he began to notice his palpitations. He now does not notice dyspnœa except on running upstairs, and then no more than he thinks anybody would who was soft and out of training. In spring of 1900 he consulted me, but nothing positive was discovered to explain his palpitations, and he was advised to give up his tobacco. This he did, and his palpitations disappeared. Upon attempting to smoke again a few months subsequently his symptoms returned and he again abandoned his smoking permanently, but his palpitations still continued.

Examination of his urine two years ago showed it to be normal. Last spring he observed for the first time that a prolonged and severe attack of palpitation was followed by the passage of a large amount of pale urine. His digestion is not good, he thinks, there being a "rumbling and roaring" in the bowels, some eructations, and occasionally heart-burn. Sometimes his flatulence is followed by diarrhœa, but ordinarily his bowels are regular. His sleep is good and his habits are now excellent.

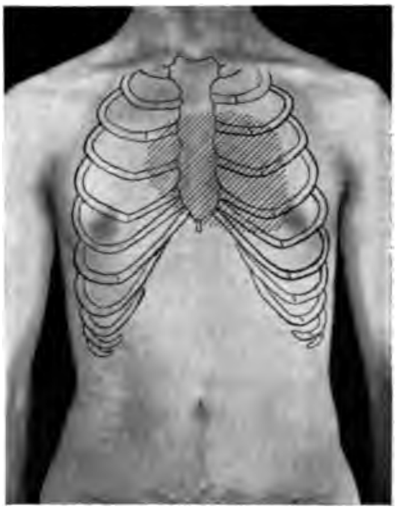


FIG. 36.—RELATIVE DULNESS. CASE OF MITRAL INSUFFICIENCY.

The pulse varies much in its irregularity, being steady for twenty or thirty seconds, and then intermitting every few beats; it is equal, of good volume, and of normal rate. There is no perceptible cardiac impulse except when the heart gives a more than usually vigorous contraction. Absolute dulness is normal, but relative dulness measures 1½ inches to right of the sternum and 4½ to the left of the median line in the third interspace (Fig. 36). The sounds are distinct, but the pulmonic second markedly accentuated. At the apex and round about the nipple there is a faint, soft systolic murmur, which is heard distinctly only when the heart makes a strong contraction after an intermission. In the fifth interspace, within the left nipple-line, there is a suggestion of a very short presystolic murmur. The systolic murmur is increased somewhat in the recumbent position.

There can be no doubt of the existence of a mitral leak, but it is not quite clear whether it is of rheumatic origin or resulted from a degree of cardiac overstrain during that game of football, and from which the heart has not recovered.

The effect of tobacco and emotional excitement on this patient is interesting. After having both smoked and chewed for seventeen years he has at length been compelled to abandon its use, because it now invariably induces an attack of palpitation, which is described as a " fluttering." He notices also that the excitement attending a quiz or examination in class will set his heart to fluttering. Also an attack is pretty sure to come on about an hour and a half after a meal, whereas if he misses a meal the palpitation does not occur until the usual length of time has elapsed after the next meal. This individual is highly nervous, not being able to keep still, continually moving a hand or foot. It is this instability of the nervous system that accounts for the readiness with which his heart responds to stimuli that do not disturb cardiac action in most individuals.

In contrast thereto is the patient's statement that even vigorous effort does not produce palpitation and only a moderate degree of breathlessness. Lastly, he is not conscious of the habitually intermittent action of his heart when he is at rest.

The treatment advised was a blue pill once in two weeks, to be followed by a dose of salts next morning, tincture of fat-free digitalis in 5-drop doses, and $\frac{1}{30}$ of strychnine thrice daily, the avoidance of immoderate physical effort, and a dietary rich in proteids and containing a restricted amount of carbohydrates.

Intense cardiac pain of the kind to merit the term angina pectoris is rare in mitral regurgitation, particularly in the young, or when of endocarditic origin. In some, however, there are neuralgic pains above the præcordium, which, by reason of an associated sense of oppression, may be called not inaptly anginoid pains. The pain most frequently complained of by these patients is a left inframammary neuralgia, which is variously described, as if a knife were being thrust through the heart, or as if the pain would take the breath away, or as if the heart were being clutched or twitched or screwed together. This pain is apt to appear suddenly, to be sharp and lancinating, to last a few seconds, and recur after varying intervals, to be confined to the præcordia, or to radiate around the back, down into the hypochondrium, up into the neck, or even into the left arm. Occasionally the cutting pain subsides, leaving a feeling of soreness behind. This heart-pain, as it is called, is not angina pectoris, but a true intercostal neuralgia, and is very apt to be associated with palpitations. For this reason the patients are all the more convinced that the pain is in the heart itself. I have known strong men, and even physicians, dreadfully alarmed by such an attack. Its association with certain tender areas pointed out by Head indicate its origin in disorders of indigestion, and hence its frequent occurrence in patients with mitral disease. It is not to be supposed, however, that it is at all peculiar to this class of cardiopaths.

Not long ago I was consulted by a woman of thirty-six on account of a sharp pain in the left side near the heart. She gave a history of inflammatory rheumatism eighteen years before, at which time she was ill two or three months. She remembered having had scarlatina in childhood, followed by dropsy for an indefinite period. She had had two attacks of measles, once in childhood, and again at the age of twenty. Thirteen years prior to my examination she had suffered from la grippe. She had been married sixteen years, given birth to one child, and for many years had been a hard-working, active woman. Her chief complaint was a sharp pain about the heart, and yet in response to query concerning shortness of breath said that when she ran upstairs, as was her wont in order to get up quickly, she was so out of breath that she had to sit down to recover breath. At such times she also became blue. Upon rising suddenly she felt dizzy, her hands and feet swelled sometimes, was annoyed by gas in the stomach and bowels, and had a poor appetite. Bowel movements and menses were regular, though the last were scanty. She sometimes heard a grating noise that seemed to come from the heart.

Her pulse, while sitting, was 80, small, weak, and regular. The apex-beat in the fifth left interspace, nipple-line, was weak, but without thrill. Absolute cardiac dulness was normal, but relative dulness extended to the sixth costal cartilage below, 4 inches to the left of midsternum, not increased to right (Fig. 37). The first sound at the apex was feeble, being partially obscured by a murmur and succeeded by a feeble second tone, while the pulmonic second was distinctly accentuated. All over the cardiac area was a loud, sawing murmur of systolic rhythm, most distinct at the apex, and transmitted around the left side to the back. The liver was barely palpable, yet tender in the region of the gall-bladder, but in other respects the abdomen was negative. The condition was plainly a mitral regurgitation of rheumatic origin, with imperfect compensation because of the daily strain to which the heart was subjected.

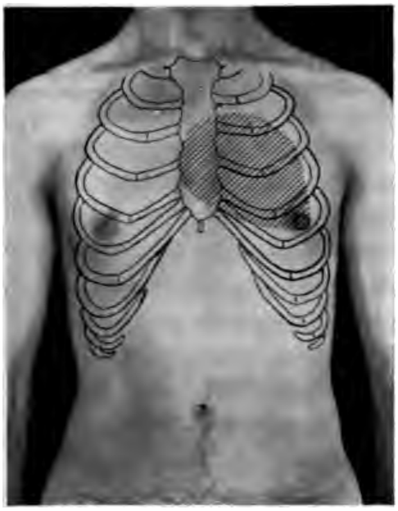


FIG. 37.—APEX-BEAT AND RELATIVE DULNESS, CASE OF MITRAL REGURGITATION (p. 229).

This case is interesting because of the fact that although the patient admitted dyspnœa and cyanosis upon unusual physical effort, she was yet chiefly disturbed by the præcordial pain. This was undoubtedly an intercostal neuralgia that was in reality due to her gastric and intestinal fermentation and only indirectly to the valvular lesion.

The only symptom of which one of my male patients complains is an intermittent pulse that annoys him whenever he breaks away from his strict diet or confines himself too closely to his office. He is always benefited by outdoor exercise, particularly fishing, and declares he has no shortness of breath and no uncomfortable palpitation unless his exercise is too violent. Another patient, who has pronounced yet perfectly compensated mitral regurgitation, is able to endure any form of exercise, even running, without any other symptom than a rapid, strong heart-action; while still a third bears without any apparently injurious effect broadsword practice and sparring.

Between such cases as these and those in the last stages of cardiac incompetence there are all grades of sufferers. In what may be called an *intermediate stage*—that is, when compensation is no longer complete—the symptom most commonly experienced is dyspnœa. Some patients do not notice this breathlessness during ordinary walking, but only when they hurry or ascend stairs rapidly. Even then they sometimes say in response to queries concerning their ability to endure exercise, they do not think they get any more out of breath than does anybody in hurrying upstairs. The fact is, such persons have been so accustomed to breathlessness, even for years perhaps, that they do not attach any importance to it, and do not consider it a symptom of heart-weakness. Indeed, so long as dyspnœa is not more pronounced, the mitral lesion may be considered in a state of fair though not perfect compensation.

In this intermediate stage there is a degree of chronic pulmonary congestion which renders these patients particularly liable to coughs and colds. These may be transient and troublesome only during damp, cold weather, disappearing altogether in summer, but in not a few cases there is a very obstinate chronic bronchitis. When this last is present, it possesses no peculiar features that distinguish it from the chronic bronchial catarrh so often observed in persons without valvular disease. It has been my experience, however, that a persistent and frequent cough is very apt to intensify the already existing dyspnœa by reason of the strain it puts upon the right ventricle. Consequently, whenever cough makes its appearance in a case of mitral regurgitation it is not to be regarded as of no importance and likely to "wear off." It may gradually wear away if the season is propitious, but it is far more likely to run on into a chronic condition. It should be borne in mind, also, that in these mitral patients cough may be attended by blood-spitting from rupture of a congested capillary in the bronchial mucosa, and is not at all significant of tuberculosis. Occasionally cough is induced by unusual exertion or excitement, and is, of course, due to the irritation of excessive pulmonary hyperæmia. In two instances coming under my notice it was attended by a profuse serous frothy expectoration that betokened acute œdema of the lungs. In both these cases the attack subsided without treatment after the patients had betaken themselves to absolute physical repose. Such attacks are not without danger, and often necessitate energetic treatment.

The following case is an example of the occurrence of hæmoptysis in mitral regurgitation. In March, 1901, a tall, slender girl of nine was brought to me by her parents, who were in a state of great alarm because of her having spit up a little blood a few days previously. One of the child's maternal uncles was in Colorado on account of his lungs, and consequently it was feared the little patient might be developing pulmonary tuberculosis. It was ascertained that there had been no antecedent cough, but that the hæmoptysis, which had occurred more than once, had followed upon rather more than the usual amount of running. There was a history of measles, but not of scarlet fever and not of rheumatism, although the child had in earlier years suffered a good deal from pains in her legs, and been wont to cry out in her sleep from night terrors. Upon examination I readily discovered a loud, blowing systolic apex-murmur that was propagated to the back. The heart was unmistakably enlarged and the liver was palpable. As a result of these findings and in the light of the history I had no hesitation in pronouncing the case one of mitral regurgitation, probably of rheumatic origin, and also in explaining to the parents that the symptom causing their alarm did not denote tuberculosis. They were told that it was the result of congestion of the lungs brought on by a degree of physical effort that was too much for her damaged heart. It may be added that the knowledge of their child's valvular lesion did not serve to allay their apprehension. This case also illustrates how remarkably unconscious of dyspnœa children often are who have mitral insufficiency, for this child not only was fond of running and playing hard, but said such sports did not make her feel bad or get out of breath.

In cases of *imperfect compensation* there are likely to be symptoms of stasis within the vessels of the abdomen as well as of the lungs. Disorders of appetite and digestion are now quite common. Some patients lose their appetite altogether, or if they feel hungry when sitting down to eat, soon find themselves full and uncomfortable, not because of having taken too much, but on account of the formation of gas that distends the stomach and produces a sense of repletion with, in some cases, an intensification of the already existing dyspnœa. These patients declare they bloat up after meals, and often complain of annoying eructations. The escaping gas is either tasteless and odourless or tastes strongly of some of the ingesta. Others suffer from acid indigestion, pyrosis, burning or gnawing in the pit of the stomach, colicky pains, etc. Some patients are constipated and annoyed by flatulency, while others have a tendency to diarrhœa, particularly in the morning before breakfast. In a few instances I have known a veritable bulimia, which it seemed to me might reasonably be attributed to the irritation of fatty acids and gases. It is not uncommon for these patients to be tormented by thirst, which, compelling them to drink largely and often of cold water, aggravates their trouble. The condition of chronic gastric catarrh responsible for these symptoms is *not peculiar to mitral patients;* it is only one of the many manifestations of secondary visceral congestion which bring the patients to the physician. The digestive defect is often the result of dietetic indiscretions, yet their mitral leak predisposes them to suffer from errors in diet which would not affect them were they healthy.

In females chronic hyperæmia of the pelvic organs is shown by leucorrhœa and menstrual derangements. In some menstruation is profuse and irregular, while in others again the catamenia are scanty or suppressed.

Stasis in the hæmorrhoidal plexuses may lead to piles and consequent constipation. Renal congestion is shown by scanty, light-coloured urine, rich in urates. Albumin and casts do not appear as a rule until in the latter stage of the valvular disease, when dropsy has come on.

Examination of the liver in this intermediate stage of defective compensation may or may not disclose enlargement of the organ. In most instances hepatic dulness is found to reach a finger's breadth or so below the inferior margin of the ribs, while the lower border of the organ feels smooth, rounded, and firm. Palpation may also disclose more or less tenderness of the organ, so that the patient winces and involuntarily resists further palpation. If the congestion has affected the biliary passages and led to their catarrhal occlusion, more or less complete, this may be shown by icterus. As a rule this is slight, and combined with capillary congestion produces a muddy hue of the face.

In the most of these cases of partially destroyed compensation there is not actual cyanosis, but instead the lips and cheeks present a dark-red colour that by the uninitiated is very apt to be mistaken for an appearance of ruddy health. If the hands and arms are inspected their superficial veins are seen to stand forth too prominently, while by night the ankles display, if not actual pitting, at least a degree of puffiness and tension which is but a little way removed from œdema and betokens marked capillary stasis.

The patients now find their breath uncomfortably short on hurry, and even when they walk leisurely they are conscious of slight breathlessness. In addition, they notice a feeling of fulness or tightness in the heart-region, which if not actually painful is very akin to pain. They find also that they become fatigued more easily than formerly, and that talking requires more effort than is quite comfortable, while if they converse during walking they pant noticeably. Efforts before endured without conscious effect now throw them into perspiration, and make them glad to sit or perhaps lie down and rest. Such symptoms constitute the earliest evidence of cardiac inadequacy, and if not heeded will go on to symptoms of positive loss of compensation.

In some persons in this stage there is marked tendency to drowsiness after meals or whenever they sit quiet and strive to fix their attention on a speaker. This is especially noticeable if the atmosphere is close and hot. Some persons find the heat and closeness produce a feeling of faintness or suffocation which necessitates their seeking the open air. Sleep is heavy, with frightful dreams, or there is insomnia, so that in the morning the patients are intensely weary and unrefreshed. Headaches are not uncommon in this stage, and for the most part are dull frontal pains accompanied by a sense of mental weariness and confusion.

A still more advanced stage of lost compensation is shown by a young lady with free mitral regurgitation and adherent pericardium whom I have under observation at this present writing. The skin along the shaft of each tibia can be indented slightly by firm pressure; the external jugulars stand forth as large as my little finger, are painful, and show the positive venous pulse of tricuspid leakage; the waist looks and feels disproportionately large, while below the ribs the big resisting liver extends to the level of the umbilicus, causing the abdomen at its upper zone to stand out unnaturally; the veins on the hands and arms are turgid, while the pulse is small, weak, and faltering, but not much accelerated, seldom reaching 100. Examination shows a diffused unsteady impulse throughout the præcordium, while the broad, rather strong, immovably fixed apex-beat is in the sixth interspace, almost to the left anterior axillary line; absolute cardiac dulness is so enormously increased both by reason of dilatation and the retraction of the lung-borders, as to nearly correspond with relative dulness. All over the left chest can be heard an intense systolic murmur, which is unmistakably a mitral regurgitant one, while to the right of the lower portion of the sternum is a somewhat softer systolic bruit that is probably tricuspid. The scanty urine contains 6 per cent of albumin, hyaline and granular casts, while menses have been absent for the last six months. In the way of subjective symptoms there are the following: Shortness of breath when walking or conversing, an occasional short, dry cough, pain in the distended jugulars, when these are unusually full, a sensation of fulness and tightness in the region of the liver, at times an intense uncontrollable nervousness and restlessness, but almost no sense of digestive discomfort, and the appetite is remarkably good.

This patient's first breakdown occurred ten years ago, and has been followed by two or three others. This present manifestation of ruptured compensation began six months ago, and for several months was so bad that she had ascites, dropsy of the ankles, and was confined to her apartment. She was not then under my care, but was at her home in an adjoining State. In this case there are to me two features of particular interest: (1) The complication of pericardial adhesions, which utterly preclude the possibility of reducing the dilatation of the left ventricle by which the regurgitation is intensified, and (2) the absence of dropsy, although the tricuspid-valve is incompetent. Compensation is destroyed, and I fear irretrievably so, but the disturbance of circulation has not yet reached the degree nor produced the distressing subjective symptoms sometimes witnessed in cases of mitral regurgitation.

When in this disease *compensation is wholly gone*, there is a marked aggravation of all the objective and subjective symptoms that have been described. There are now evidences of extreme stasis. Dropsy is generally a pronounced feature, and in many cases dominates the scene. Beginning at the ankles, it creeps upward until it involves the integument of the trunk, and it may be of the upper extremities. There is also in this stage transudation into the serous cavities, ascites, hydrothorax, and pulmonary œdema. This condition leads to orthopnœa, cough, and frothy or even bloody sputum. I recall a female who in this condition of hopeless suffering was sitting on her bed, supporting her elbows on her knees and her face in her hands, and was coughing pure blood. She had been in this plight for days and had abandoned hope, having been told by more than one doctor that her condition was hopeless. Three months thereafter she was walking about the house free from œdema. This case is described more fully in the chapter on Treatment. Another female patient who now visits my office was, four years ago, so dropsical that she appeared waterlogged, and for six weeks had been obliged to get what sleep she could in an easy chair and resting her arms on a table in front of her.

When stasis reaches such an extreme degree as in this last case it has generally been preceded by dilatation of the right ventricle and signs of tricuspid insufficiency. This condition of the right heart may precede and seem to usher in dropsy, or serous transudation may anticipate the appearance of relative tricuspid incompetence. There is such a difference in the extent of anasarca, and in the stage of the disease at which it begins to develop, that there is something more than mere venous stasis to account for it. Accordingly, it is now known that serous transudation takes place when, in addition to stasis in the venous system, there is an increased permeability of the capillary walls due to disturbed nutrition, as well as a condition of hydræmia.

In some cases dropsy becomes so excessive that the skin of the legs becomes red and shining, or even forms blebs, which, bursting, permit the serum to ooze forth, and thus diminish the tension of the surrounding parts. Under such circumstances the nutrition of the integument becomes so impaired and infection is so easy that a simple scratch or abrasion may set up inflammation.

In this stage of things extreme gastro-intestinal congestion and abdominal distention from fluid and flatus prevent the taking of adequate nourishment. Cerebral congestion and œdema produce headache, insomnia, or somnolence, an unreasonable fretfulness and irritability, or a low, muttering delirium. The patient's condition is now one of indescribable and unendurable suffering, so that physician and friends alike breathe a sigh of relief when, after weeks or months of such hardship, death ends the struggle.

It only remains to say a word concerning those cases of mitral regurgitation which are either complicated by chronic nephritis or are secondary to the dilatation of the left ventricle consequent upon the Bright's disease. These cases do not differ essentially from those of the class just considered. There is, however, an element of uræmia in these cases which is apt to modify the picture somewhat. These patients are apt to suffer from a form of dyspnœa that is very distressing and difficult to relieve by treatment. It consists of sudden paroxysms or intensifications of their habitual shortness of breath which come on independently of exertion or any other exciting cause. These may be worse at night—are so generally, but are not necessarily so, for I have known them to occur by day. They have always seemed to me to be of toxic origin or to be partly renal and partly cardiac. Headache and nausea are also likely to torment the patient, and inflammations of the serous membranes are not uncommon. Dropsy is often very pronounced, and is peculiarly soft and rebellious to treatment. In other cases pulmonary and cerebral symptoms predominate. They all furnish an absolutely unfavourable prognosis on account of the associated or antecedent kidney lesion.

Paroxysms of dyspnœa, sufficient to be termed cardiac asthma, are not very common in mitral incompetence, unless some complication, such as chronic nephritis, is present, to which the regurgitation is sometimes secondary. In these latter cases asthmatic seizures are frequent, or the dyspnœa assumes the Cheyne-Stokes type. Likewise, other symptoms properly belonging to mitral regurgitation may, when associated with chronic nephritis, be modified by uræmic manifestations. There may be very distressing nausea and vomiting, and the physician may be at a loss to know whether the symptoms be due to renal inadequacy, or to passive congestion of the stomach depending upon the regurgitation.

If in a given case of mitral regurgitation ascites appears prior to or independent of anasarca, it is due to some complication, and in my experience this has most frequently proved to be adherent pericardium. These are the cases sometimes described as pseudo-atrophic cirrhosis of the liver.

Embolism from the detachment of a vegetation is not common in mitral insufficiency unless it be complicated with acute endocarditis, yet this untoward event may occur, and then produces symptoms depending upon the organ in which the embolus is arrested. If this be the kidney, there is likely to be bloody urine, and yet it is not uncommon to discover post-mortem evidences of infarcts produced by emboli of such minute size as to have escaped detection during life.

Cerebral embolism produces symptoms too characteristic to escape notice. As the left middle cerebral artery is the one most commonly plugged, it is followed by aphasia and right-sided hemiplegia. An embolus entering the hepatic artery produces acute icterus and symptoms closely resembling acute yellow atrophy of the liver. In the case of the spleen, the sudden, sharp pain is likely to be followed by tenderness and enlargement of the organ. If an artery of one of the extremities is thus occluded, characteristic pain is followed by loss of pulsation in the artery below the seat of embolism, by weakness, numbness, coldness, and paræsthesia, and even by gangrene of the limb, if the main artery happens to be plugged. Usually, however, these symptoms gradually disappear with the establishment of adequate collateral circulation. A by no means uncommon effect of the terminal stage of mitral regurgitation is the occurrence of pulmonary infarcts due to the lodgment in the pulmonary capillaries of minute fragments of thrombi that have formed in the dilated right chambers of the heart. Pain in the affected lung may or may not be felt, but if the patient suddenly begins to cough up clear blood it is very suspicious of pulmonary infarction.

Very exceptionally, a mass of considerable size may be swept off from the cardiac thrombus, and entering the pulmonary artery or one of its large branches, may produce instantaneous dyspnœa and speedy death.

Physical Signs.—*Inspection.*—There is nothing in the appearance of individuals with a latent mitral insufficiency to suggest the existence of their disease. In some instances capillary congestion is just sufficient to impart to the lips and cheeks a slightly heightened colour that is easily mistaken for the hue of health; it is, however, deeper than the rosy glow of perfect circulation. In a still more advanced degree of congestion the lips become of a bluish or even purplish colour, the capillaries of the face are injected, and the fingers also display more or less cyanosis. There is, however, nothing in this appearance to indicate more than impeded circulation, and hence it is not peculiar to mitral regurgitation.

In children with long-standing mitral disease the fingers are apt to be clubbed, the stature stunted, the shoulders stooping, and the præcordium bulging. In adults, on the other hand, inspection of the cardiac area reveals no alteration of the kind seen in children.

The apex may be displaced somewhat to the left, depending on the degree of left-ventricle hypertrophy, and if serious dilatation is not present is broader and stronger than in health. If the right ventricle is also sufficiently enlarged to lie well down in the sulcus formed by the union of the diaphragm with the anterior chest-wall, its pulsations are seen more or less distinctly directly below the ensiform appendix. This *epigastric pulsation forms an important sign* of right-ventricle hypertrophy, and hence is a secondary sign of mitral disease. It should be carefully distinguished from the throbbing of the abdominal aorta, so often observed in this situation in neurotic individuals with thin abdominal parietes. Of course the information to be derived from inspection depends largely upon the condition of the thoracic walls, and hence inspection is of greatest value in persons whose hearts are disproportionately large as compared with the size of the chest.

Palpation.—In compensated and uncomplicated mitral regurgitation the pulse possesses no distinctive characters aside from its lowness of tension. Its rate is usually somewhat accelerated, its tension low, and in compensated cases at least, it is regular in frequency, force, and volume. As the energy of the heart wanes, as its walls become degenerated and its auricles dilated, the pulse grows strikingly irregular and intermittent, the pulse-waves differing from each other in size and strength, coming at uneven distances, and often dropping out altogether even when the heart-beats are not themselves intermittent (Fig. 38). Such a pulse is exceedingly difficult to count, and if subjected to a little pressure by the finger, for the purpose of having its characters brought out more distinctly, it disappears from beneath the palpating finger in a manner that makes it very elusive. This extreme irregularity of the pulse is most frequent when regurgitation is combined with stenosis or an adherent pericardium.

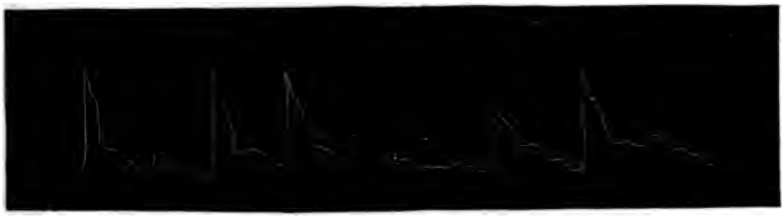


FIG. 38.—SPHYGMOGRAM OF CASE OF MITRAL REGURGITATION, SHOWING IRREGULARITY OF PULSE.
(Personal observation.) Enlarged.

The pulse in the two wrists is generally equal, but cases have been described in which the right was distinctly smaller than the left, owing to relative tricuspid insufficiency and consequent pressure of the dilated right auricle on the subclavian artery. Balfour states that if the arm is elevated the peculiar characters of the pulse—i. e., its *irregularity and lowness of tension*—are brought out more distinctly.

Palpation of the præcordia confirms the information obtained by the eye, but in addition enables one to better appreciate the strength of cardiac contractions. It may also detect thrill or pulsations that cannot be perceived by inspection. Palpation is often a valuable means of ascertaining the size of the heart, particularly in females in whom excessive mammary development may prevent accurate percussion. By pressing the fingers gently yet firmly into the intercostal spaces beneath the breast one can ascertain the position of the apex-beat, and thus judge of the size of the left ventricle.

Authorities differ as regards the existence of a thrill at the apex in cases of pure and uncomplicated mitral regurgitation. When, however, such a thrill is present it is systolic, and felt with greatest intensity at the immediate seat of apex-impulse, since it is the palpable expression of the murmur. For my part I am perfectly sure of its occasional existence in cases without conjoined mitral stenosis.

Lastly, by palpation of the epigastric notch one can also judge of the degree of compensatory enlargement and vigour of the right ventricle. In this way one can often determine that the ventricle is hypertrophied, when for one reason or another it is difficult or impossible to outline the chamber by percussion.

Percussion.—This means of cardiac examination should never be neglected, since as a general thing it furnishes the most valuable and reliable information concerning the heart's condition. Indeed, percussion is almost our only means of ascertaining the size and shape of the heart, and hence of learning what and how extensive have been the secondary changes wrought by the valvular defect. If in the lesion now under consideration percussion does not detect increase of absolute or relative dulness to the right and downward, the inference is warranted, even though there be an intense systolic bruit, that the leak is not free, or that being free it has nevertheless been perfectly compensated. In most cases of mitral regurgitation, however, the lesion has led to enlargement of the right ventricle, and in such an event, deep-seated if not superficial dulness is increased to the right and inferiorly; and hence the extent to which dulness is increased is a criterion by which we can judge of the freedom of the leak or of the compensation. Secondary enlargement of the left ventricle is shown by increased dulness of the left (Fig. 39).

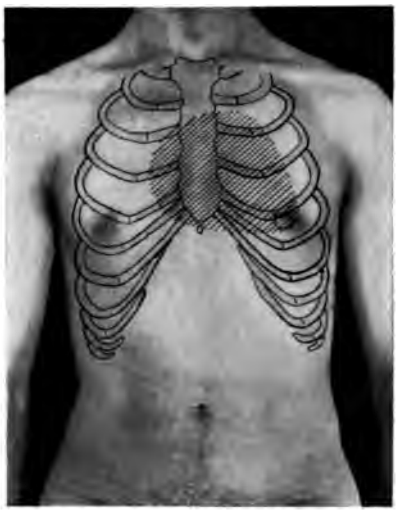


FIG. 39.—RELATIVE DULNESS IN A TYPICAL CASE OF MITRAL REGURGITATION.

Mitral regurgitation, it will be remembered, leads to dilatation of the left auricle as well as hypertrophy; hence in pronounced cases the outline of the deep cardiac dulness at its upper and outer corner, so to speak, is broad and rounded, corresponding to the enlargement of the auricle. It is not always easy to determine this alteration of shape by percussion; yet if firm percussion is made, and the chest-wall is thin and yielding, it is sometimes possible to determine the extent to which the left auricle has been affected by the regurgitation.

Auscultation.—This forms a very valuable means of cardiac examination, for without the information thus obtained one cannot safely assert that mitral regurgitation does or does not exist. *It should not be relied upon to the exclusion of other methods of investigation,* however, for reasons that will be stated presently. The auscultatory evidence of valvular disease lies in certain acoustic phenomena which are produced by soniferous currents in the blood-stream, and are called murmurs. It is plain that many different factors influence the character of a murmur, and that if reliable information is to be derived from the study of a murmur the characters peculiar to each must be understood.

The auscultatory indication of mitral regurgitation, then, is a systolic murmur heard with maximum intensity in the mitral area—i. e., at or close to the apex-beat (Fig. 40). Such a bruit is not, however, an invariable sign of mitral incompetence, since it may be accidental, and hence there are other facts concerning a mitral regurgitant murmur which must be understood.

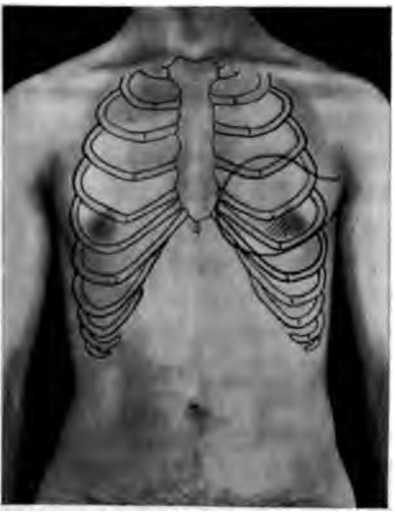


FIG. 40.—POINT OF MAXIMUM AUDIBILITY (SHADED) AND AREA OF TRANSMISSION (OUTLINED) OF MITRAL REGURGITANT MURMUR.

The murmur is systolic because produced during the contraction of the ventricles, and therefore it is strictly synchronous with the first sound, although it not infrequently lasts a little longer, and may even persist and increase in distinctness through the short pause up to the succeeding second sound. It is so common for some degree of obstruction to be combined with regurgitation that a shorter or longer presystolic murmur often ushers in the systolic bruit. It forms but an added element, and in nowise alters the fact stated above—namely, that the ***time or rhythm of the mitral regurgitant murmur is strictly systolic*** (Fig. 41).

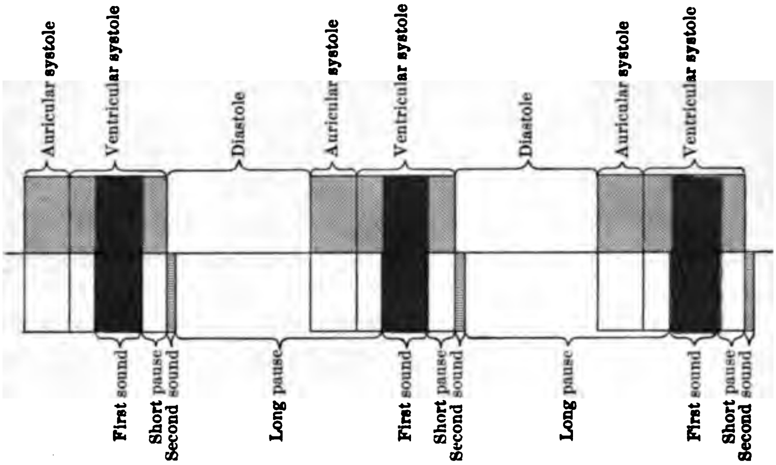


FIG. 41.—TIME OF MITRAL REGURGITANT MURMUR.
Red shows time of murmur. Cardiac cycle as in Fig. 9. Read from left to right.

The next element of importance is its position of maximum intensity. The bruit in some cases is heard most clearly near the anatomic situation of the mitral valve—i. e., at the level of the fourth costal cartilage near the left border of the sternum, but as a general rule the murmur is conducted along the chest-wall to the point where the apex of the heart strikes with greatest force. Hence the murmur is heard most loudly in the immediate vicinity of the apex-beat, sometimes slightly within, sometimes just above the nipple, and sometimes a little to the outer side of the apex. It is, however, louder in this than in any other cardiac area, and by reason of this circumstance recognised as mitral.

Very rarely a mitral regurgitant bruit is heard more plainly in the tricuspid area—i. e., on the ensiform appendix or close to its left margin—when it is likely to be mistaken for a tricuspid bruit. I have known such a mistake to be made in more than one instance. The error can be avoided by attention to the signs of tricuspid regurgitation, as described in that chapter.

Inexperienced auscultators are apt to attach a wrong importance to the intensity and the quality of a murmur. It goes without saying that all possible differences in these respects are to be found in mitral systolic murmurs depending upon conditions governing their generation. Neither the loudness nor the quality of a bruit furnishes any evidence *per se* as to the gravity of the lesion. A very intense musical murmur may be produced by the blood-stream being forcibly driven through a small aperture, and conversely a very widely open and unguarded orifice may permit the blood to regurgitate so easily and noiselessly that only a very soft, scarcely audible murmur is generated. Hence neither intensity nor quality of a murmur is of importance in determining whether or not it is mitral; they only facilitate the detection of the murmur, and sometimes aid us in determining the seriousness of the lesion.

Furthermore, the intensity of the murmur is governed by other circumstances than the leak itself. The bruit of mitral regurgitation is generally loudest during energetic action of the heart, hence during excitement and immediately after exertion. It is consequently brought out clearly by having the patient jump about, swing the arms violently, or do something else that causes the heart to beat rapidly and energetically. By such a procedure it is often possible to detect a mitral bruit which before was inaudible or so indefinite as to have left the examiner in doubt of its existence.

The position of the patient's body also influences the audibility of the murmur. It is in most cases heard most plainly when the patient sits or stands, but I have frequently seen cases in which it came out *far more distinctly in the dorsal decubitus*, which permitted the heart to beat more forcibly because more slowly. Consequently, it should be an invariable rule to auscultate a suspected case of mitral insufficiency, and indeed any suspected case of cardiac disease, in all three positions. It will often protect one against a serious blunder in diagnosis.

Mitral systolic murmurs are usually spoken of as blowing and soft. They are as a matter of fact softer than direct murmurs of stenosis, but they are by no means always soft. They may be harsh and rasping, or filing, grating, sawing, whistling, etc., in which event they are designated as musical, a character of pathological but scarcely diagnostic interest.

Finally, mitral systolic murmurs should always be studied with respect to the direction in which they are transmitted from the apex. This is especially important in cases in which secondary physical signs of mitral regurgitation are difficult to obtain, for in such cases it is necessary for correct diagnosis to determine whether or not the apex systolic bruit is an accidental one. Such inorganic murmurs are of limited propagation, whereas mitral regurgitant bruits are often, though not invariably, widely transmitted. Their direction of propagation is towards the left rather than the right, and therefore they may be traced into the axillary region, or, as sometimes happens, even on to the back. It is not at all rare to hear an intense mitral murmur at the inner side of the left scapula near its tip, and in children such a bruit may be heard throughout the entire thorax. When in any case the murmur is audible in more than one area it is indispensable to determine by careful comparison in which area it is of maximum intensity, for only in this way can one decide to what cardiac area the murmur belongs.

One is not to content himself with studying only the murmur, he must also *carefully auscultate the several heart-sounds.* If the first tone at the apex is not replaced by the murmur, it offers a certain amount of evidence in favour of the valve being not wholly destroyed, but able to still partially close the orifice. If, on the other hand, the murmur alone is heard, it indicates great freedom of regurgitation. Then one should note the degree of accentuation and purity of the pulmonic second sound, especially in all cases in which the interpretation of the murmur is not clear. Regurgitation by inducing pulmonary congestion leads to *intensification of the pulmonic second tone*, and hence such intensification is the earliest recognisable secondary physical sign of mitral insufficiency, and greatly strengthens the inference drawn from the recognition of a murmur.

Diagnosis.—The diagnosis of mitral regurgitation is not difficult as a rule, being in some instances one of the easiest of all valvular lesions to make out. When, however, the leak is slight, the murmur obscure, and the secondary changes in the heart and general circulation insignificant, its diagnosis may be anything but easy. It is also occasionally difficult when there is dropsy, a rapid, tumultuous action of the heart, extensive dilatation, and serous accumulation in the pleural cavities. One may then make a diagnosis of the condition with reservation, and wait for treatment to clear up the case. Attention to the secondary physical signs will usually help out amazingly under such circumstances.

The history of the patient is also of great importance, not alone to the diagnosis of mitral insufficiency, but to the recognition of the etiology, and thereby to the relative or organic nature of the regurgitation. Its gravity is to be determined by the secondary effects which the heart-walls and cavities have undergone, and by the presence or absence of symptoms referable to the cardiac disease. Not until all this has been accomplished should the physician rest satisfied, remembering that the *detection of a murmur does not constitute a diagnosis.*

Prognosis.—In general, it may be stated that mitral regurgitation affords a more favourable prognosis than does any other valvular disease, yet in this respect each case is a law unto itself. Furthermore, the prognosis of each case depends upon many different factors: (1) On the etiological nature of the defect, (2) its severity, (3) the degree of secondary effects, (4) the completeness of compensation, and (5) the existence or not of complications, such as other valvular lesions and adherent pericardium.

Insufficiency of the mitral valve from *sclerosis* furnishes as a rule less favourable prognosis than does that of endocarditis, because of the progressive tendency of the sclerotic change and of the age of the patient, which renders it likely that the myocardium is no longer healthy. If the regurgitation is free, if secondary hypertrophy and dilatation are extensive, if engorgement of the general viscera is apparent, even though compensation seems adequate, the prospect of the disease remaining stationary is not good. If mitral regurgitation is united with defects of other valves or orifices, the prognosis is correspondingly unfavourable. Complications on the part of the pericardium and of chronic nephritis render prognosis very unfavourable. Finally, prognosis *stands in direct relation to the completeness of the compensation.*

The curability of this lesion resulting from endocarditis has been much discussed, and, as pointed out by Balfour and others, seems certainly possible. This is considered particularly true after chorea.

The influence of age, sex, occupation, environment, etc., will be considered in a subsequent chapter devoted to the Prognosis of Valvular Disease in General.

Mode and Causes of Death.—A patient with uncomplicated mitral incompetence *rarely dies suddenly* and without warning, as in some other forms of heart-disease. When, however, this lesion is united with fatty or fibroid degeneration of the heart-muscle the individual may drop dead from sudden diastolic arrest. I have known one such instance in a man of sixty. A long period of suffering may be terminated by a rather rapidly developed pulmonary œdema, but usually the end comes slowly through gradually increasing cardiac exhaustion and weeks or even months of most distressing symptoms.

Very rarely, death may take place from embolic plugging of the pulmonary artery or one of its main branches, but such a sequence is usually preceded by symptoms of cardiac asthenia.

In one instance a young woman died suddenly under the following circumstances: About a week before her death she had sought medical advice on account of increasing dyspnœa, and was found to present signs of combined mitral incompetence and obstruction due to articular rheumatism some years before. Because of failing compensation she was ordered to rest quietly at home; notwithstanding this, she a few days later carried a bucketful of coal upstairs. The next morning her speech was thick, she complained of stiffness in the back of the neck, and showed a degree or so of temperature, but evinced no paralysis. She was then sent to the hospital, and a day or two later, upon sitting up in bed to drink a cup of tea, suddenly fell back upon the pillow and expired. A post-mortem examination could not be obtained.

Another female patient who for eleven years had evinced symptoms and physical signs of a double mitral lesion with adherent pericardium, at length began to suffer from increasingly frequent attacks that seemed to indicate a sudden augmentation of stasis within the pulmonary vessels, yet without other manifestations of more than usual cardiac weakness. Serous transudation could nowhere be detected. The final attack lasted but a few hours, and seemed to be the result of a rapidly increasing failure on the part of the right ventricle. No necropsy was permitted.

A lad of twelve years with chronic mitral regurgitation of rheumatic origin in whom a badly broken compensation had been partially restored, and who a few weeks before death seemed to have contracted a fresh inflammation of the mitral orifice, arose from bed early one morning to pass urine; he had scarcely made the attempt when he fell on his pillow in a condition that alarmed his nurse. Two hours later I found him partly unconscious, and with a moderately slow and irregular pulse. He was pronounced moribund, and death occurred an hour or two subsequently. Unfortunately, post-mortem examination could not be obtained, and as in the two preceding cases, the immediate cause of death could only be conjectured. It was probably due to an acute overdistention of the heart, leading to gradual paralysis.

Hustedt examined 491 cases of heart-disease at the Pathological Institute at Kiel, with a view to determining the causes of death. Of 15 cases of mitral insufficiency, without associated cardiac lesions, he found the following causes of death: Cardiac asthenia (Herzschwäche), 7 cases; pulmonary infarct, 2 cases; apoplexy due to embolism, 1 case, while in the other 5 death was due to some accidental or intercurrent affection, such as nephritis 1, peritonitis 2, marasmus 1, pulmonary collapse 1.

CHAPTER VII

MITRAL STENOSIS

This term denotes a narrowing or constriction of the opening between the left auricle and ventricle, in consequence of which there is an obstruction to the free flow of blood from the former into the latter chamber. A narrowing is *always the result of structural defect,* either of the ring itself, of the valve, or of both, and can never be of a functional or relative kind, analogous to relative incompetence of the valves. The stenosis may be congenital in consequence of defect of development, or of endocarditis during intra-uterine life, but in the great majority of cases it is acquired after birth, and forms one of the most frequently encountered of all valvular lesions.

Morbid Anatomy.—In a well-marked case of mitral stenosis the cusps are thickened and rigid, they are adherent, and bound firmly in place by the thickened and contracted chordæ tendineæ and papillary muscles. The whole valvular structure is thus often converted into a rigid funnel-shaped opening, with a narrow slit-like extremity of size scarcely to admit a small probe. This is the so-called *buttonhole mitral* (Fig. 42), and in this form of stenosis the endocardium may present no evidence of old vegetations, but be perfectly smooth. This has led some French authors to consider the condition one of congenital malformation rather than of rheumatic origin. Sansom, on the other hand, thinks it due to inflammation, and that its smoothness results from the "quasi-cicatricial tissue" being subjected to pressure by the blood on both its auricular and ventricular aspect.

The stenosed valves often show, however, marked evidence of past inflammation in the form of organized or calcified thrombi, especially on the auricular surface. These may be so large as to almost completely obstruct the orifice, while their presence always leads to shrinkage and deformity of the valve, and almost always to a certain amount of regurgitation. In fact a pure stenosis is very rare, although it does occasionally occur.

Campbell ingeniously suggests that the shape of the orifice determines the amount of the discharge. The shape of an orifice and the passage leading up to and away from it influence the quantity of fluid that can pass through it in a given time. "If a round

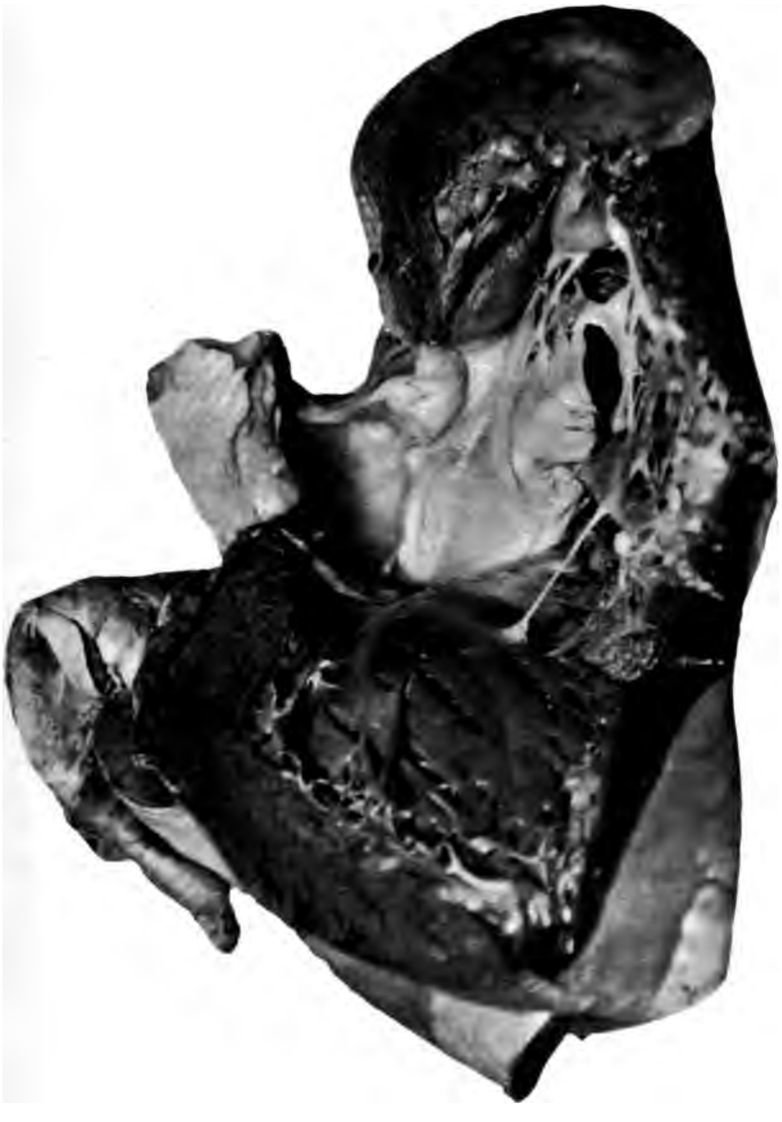


Fig. 42.—Interior of Left Ventricle, showing Buttonhole Mitral Slit. Notice aberrant tendinous cord.

hole be punctured in a can full of water, the cross-section of the fluid jet coming from it is much less than the area of the aperture, little more than half of it. The relation of one to the other is termed the coefficient of discharge. If now the small end of a funnel be accurately fitted to the inner side of the aperture, so as to imitate the condition of things obtaining in the funnel mitral, the coefficient of discharge is almost doubled. Again, if instead of making a round aperture in our can we make a linear one, so as to imitate the buttonhole mitral, we in a similar way nearly double the coefficient of discharge. This form of the mitral orifice results from the flattening out of the funnel through cicatricial contraction, so as to form a more or less flat diaphragm, and it is indeed a remarkable fact that in this process the round aperture of the funnel is invariably converted into a slit. Sometimes it will seem to be slit-shaped before the flattening-out process begins. Hence it is clear that in mitral obstruction the heart avails itself in a very cunning way of principles well known to the engineer, so as to secure the maximum flow through the narrowed mitral orifice—a brave attempt to make the best of a bad job."

It seems possible, however, that the fact of the mitral valve being composed of two approximately triangular leaflets, which become adherent along their sides, and leave a small opening at their apices, where the two flat cusps come together, may have something to do with the slit-like shape of the stenosed orifice.

The effect of narrowing of the mitral orifice is to increase the difficulty with which the left auricle expels its contents into the ventricle. The reaction of the musculature to this increased demand for work is shown by the production of hypertrophy. This is the primary effect; and dilatation, when it does occur, is a later event. In mitral incompetence, on the other hand, dilatation comes first and hypertrophy afterward. Yet there are many cases of mitral stenosis in which the auricle is found post mortem to be thin-walled and dilated. This is probably in most cases due to associated regurgitation, for in a series of cases taken from the records of Guy's Hospital, Samways found that the degree of dilatation was nearly always proportional to the amount of leakage associated with the stenosis.

The left ventricle presents a condition in marked contrast to that found in regurgitation. The narrowed orifice allows only a reduced amount of blood to pass into the ventricle with auricular systole, and hence the work required of the ventricle is reduced. The chamber becomes diminished in size, and its walls thin and weak. At times this atrophy of the ventricle is so marked that the chamber is almost rudimentary in appearance.

The effects of mitral stenosis on the circulation, pulmonic and systemic, are practically those of insufficiency, being primarily due to obstruction of the blood in the pulmonary veins. The obstruction is more constant and unyielding in stenosis, however, and hence the congestive effect is even more marked than in insufficiency.

The effect on the myocardium in producing brown atrophy, and the congestion of the various organs of the body, are the same as in mitral incompetence, and do not call for repetition.

Etiology.—Much of what has been said concerning the causation of mitral regurgitation applies equally to narrowing of the mitral ostium. There is one great difference between the two, however—namely, stenosis cannot be anything else than a structural defect, and therefore it results either from changes during fœtal existence or from endocarditis or sclerotic changes after birth. Acute inflammation usually produces clinical phenomena of incompetence and not narrowing, but changes initiated during an acute attack may subsequently develop into such as cause pronounced obstruction.

Mitral stenosis results most often, therefore, from subacute or chronic rheumatism, and on this account is a progressive lesion. Accordingly, as pointed out by Sansom, it is especially apt to be observed in persons who either give no history of acute rheumatic attacks or have suffered from vague joint pains. It is probable, therefore, that in the majority of cases mitral stenosis is of rheumatic origin, but of the insidious or masked type, and not of the pronounced form. Nevertheless, as previousy stated, a valvulitis initiated during an attack of rheumatic fever may in time eventuate in a predominating obstruction.

Another interesting view of the etiology of this lesion, and one that merits consideration, is that it has its origin in tuberculosis. This opinion was announced by Teissier as a result of his study of a large number of cases. Although positive evidence of the correctness of his views cannot be obtained, as he himself admits, he yet believes that tuberculosis lies at the bottom of the cases of mitral stenosis which are progressive.

While very loath to accept Teissier's conclusions, I am nevertheless greatly interested in the possibility of such a causative relationship, for the reason that prior to my knowledge of his views I had been struck with the fact that several times I had encountered narrowing of the mitral orifice in young women belonging to tuberculous families. I recall distinctly a young Irish girl who had lost a sister from consumption and sought my opinion and treatment because she feared she was going into a decline. The character of the respiratory murmur at the left apex made me very uneasy, and hesitate about expressing an opinion until after I had kept her under observation for a sufficient time to note any changes that might take place for better or worse. To my great surprise I at length, after repeated examinations, discovered signs that indicated a very slight and progressive mitral stenosis. After the lapse of a number of months, during which she occasionally complained of vague leg pains without other definite indications of rheumatism, a very pretty but short presystolic murmur became unmistakable.

In another woman of twenty-nine, who also had lost a brother and a sister of consumption, I found a very considerable narrowing of the mitral ostium, as evinced by the secondary signs and symptoms as well as by a long, rough, loud presystolic bruit and corresponding thrill. There were also impaired resonance and broncho-vesicular breathing at the right apex, which made me very suspicious of latent tuberculosis. In this case most careful and searching inquiry failed to elicit a history of previous rheumatism or leg pains in childhood that might have been construed as rheumatic. Nevertheless, I should be most reluctant to say that in this case the stenosis was of tuberculous origin. To my mind it is far more reasonable to assume that she had had unrecognised rheumatism. Are we to conclude because tubercle bacilli have been identified in endocarditis that these slowly progressive and often latent cases are necessarily of tuberculous causation? The natural delicacy of constitution in these individuals who come of tuberculous stock, and the frequency with which rheumatism of children is overlooked, makes far more plausible, as I take it, the conclusion that the valvular defect dates from early years of life, and has taken years to reach that degree at which it becomes clinically recognised.

Based on the teaching of Rokitansky, the view was formerly held that there is a *natural antagonism between narrowing of the mitral ostium and pulmonary tuberculosis* because of the congestion of the lungs produced by the stenosis. This is now known to be erroneous, for pulmonary tuberculosis and mitral obstruction have been observed conjointly in a considerable number of instances. Thus Sansom has collected 31 cases in which these two diseases were found associated at the necropsy. It is worthy of note also that of these 31 cases mitral stenosis and tricuspid stenosis were combined in 11, while in 5 others there was also aortic valve-disease. Sansom explains the connection of pulmonary consumption with mitral narrowing on the ground that the latter lessens the natural resistance of the organism to the inroad of tubercle bacilli. The union of aortic and mitral disease intensifies this susceptibility, while tricuspid stenosis predisposes to pulmonary tuberculosis in the same way as does obstruction at the pulmonic orifice.

Syphilis and gout are etiological factors that should not be disregarded; but they lead to the sclerotic or atheromatous, not to the endocarditic form of stenosis.

The influence of *age* upon the type of the disease is also interesting. It is the rheumatic or endocarditic form that is encountered in the young. This is shown even in the case of an infant that lived but twenty-four hours, and in whom Benezard Smith discovered mitral stenosis post mortem, and likewise in the infant of four months observed by Gerhardt, since in both these cases the stenosis was evidently the result of endocarditis during fœtal life and not of defective development. This prevalence of mitral stenosis in the young (it being detected most frequently at or about the age of fourteen, Sansom) is undoubtedly explicable by the fact that the young are most liable to articular rheumatism. On the other hand, individuals of middle age or over develop the *sclerotic form* of this valvular lesion, and in such it is probably but a manifestation of a general tendency to fibrosis.

The association of mitral stenosis with renal disease is shown by Goodhart's statistics, who found it present in about 1.4 of 192 cases of chronic nephritis that came to autopsy, while Pitt, in the post-mortem records of Guy's Hospital, found mitral stenosis and granular kidney three times as frequently as stenosis without this form of renal disease.

It is a striking fact, on which all writers comment, that mitral stenosis is encountered far more frequently in the female than in the male sex. This is especially true of the disease in persons below the age of forty, in whom it is probably of inflammatory origin, while the sclerotic form of the lesion does not appear to predominate greatly in either sex. Of 42 cases of pure mitral stenosis of which I have records, 28 occurred in females and 14 in males, and of the entire number but 20 gave a clear history of rheumatism. It is thus seen that of my cases females numbered twice as many as males, bearing out the statement that mitral obstruction is *par excellence* a disease of the gentler sex.

Symptoms.—Inasmuch as the effects of mitral regurgitation and mitral stenosis on the circulation are practically the same, there is a close similarity in the symptoms of the two affections. Therefore, much of what was said under the head of mitral regurgitation also applies to mitral stenosis. Undoubtedly this affection may remain latent for years, but it is less likely to do so than is regurgitation. Nevertheless, hard and fast lines in this regard cannot be drawn, for the manifest reason that the degree of the effect stands in direct proportion to the gravity of the lesion. If compensation is adequate, symptoms referable to the heart, or that call the attention of the patient to his heart, may be entirely absent; and yet a patient with any considerable degree of stenosis is not likely to be robust, or to possess much physical endurance.

Children are likely to be more or less stunted in development, both mentally and bodily; while in the case of adult females I have been impressed by the frequency with which they are tall and thin, with evidence of anæmia. Their circulation, as might be expected, is defective, as shown by coldness of the hands and feet and great sensitiveness to low temperature. Even when not suffering from symptoms referable to pulmonary congestion, as dyspnœa, they are apt to complain of digestive and menstrual disorders, sour stomach and scanty menstruation being particularly common. They are generally constipated and their urine is diminished in amount, of correspondingly high specific gravity, and loaded with urates.

Patients with mitral stenosis are also very prone to attacks of bronchitis, which ultimately run into chronic bronchial catarrh. They are also particularly liable to acute pulmonary œdema upon extra exertion, and in such instances the cough and hæmoptysis or frothy, perhaps blood-tinged, sputum often give rise to the fear of pulmonary tuberculosis. I have this very day seen a well-marked instance of the kind.

In other cases there is persistent dry cough due to bronchial congestion, which may attract attention from the heart to the lungs. I well remember the case of a lady who consulted me for an obstinate dry cough, which was found due to a mitral stenosis, the existence of which had not been suspected. Indeed, I myself had examined her about a year previously during an attack of tachycardia, and at that time was unable to detect any implication of the valves.

As a rule breathlessness on exertion is an *early symptom* with patients suffering from pronounced mitral constriction, even though in all other respects compensation seems good. When the narrowing of the orifice is extreme, when the heart-muscle begins to fail from degeneration or preponderating dilatation, dyspnœa becomes an exceedingly distressing symptom, and may be present, though in a less degree, even when the patient is at rest. Palpitations may also be an annoying feature, and there may be sharp or dull præcordial pains with areas sensitive to pressure, the same as in mitral regurgitation. I am unable to recall a single instance in which there was typical angina pectoris.

In other cases there is a sense of præcordial fulness or distention, particularly upon exertion. More or less vertigo declares itself upon the patient suddenly assuming the erect position, or he is annoyed by a feeling of fulness or confusion in the head. This, which is a symptom of passive cerebral congestion, often amounts to actual headache. Insomnia, disturbing dreams, and other effects of venous congestion become more and more pronounced, and the patient passes into the stage of completely destroyed compensation.

Œdema, which is at first confined to the ankles and tends to disappear over night, creeps upward into the thighs, rendering locomotion difficult and painful. Owing to the feeble, rapid, and arrhythmic action of the overdistended heart, the pulse is thready, perhaps unequal in the two wrists, intermittent, and often extremely difficult to count. This intermittence may be due to cardiac intermissions, or to such an inequality in the force of the heart's contractions that some of the blood-waves fail to reach the wrist. The hands and forearms may be cold, and the superficial veins stand out prominently in striking contrast to the emptiness of the arteries.

Pulmonary congestion declares itself by increased dyspnœa that may even amount to orthopnœa, by cough and sero-mucous or sero-sanguinolent sputum, dulness at the bases of the lungs, particularly behind, and by copious, moist râles. If the tricuspid valve gives way, permitting regurgitation into the auricle, the turgid jugulars pulsate. The liver, already swollen, perhaps tender, grows still more engorged, and likewise pulsates synchronously with the epigastric throbbing of the dilated right ventricle and the so-called positive pulse in the cervical veins. The taking of food is attended with formation of gas that distends the stomach and bowels, adding greatly to the patient's distress, and rendering adequate nourishment difficult. The sufferer frequently complains of dull or burning pain in the pit of the stomach, and is tormented by an intolerable thirst. Congestion of the head is shown by duskiness of the countenance, swimming of the head, or headache, and insomnia. In some cases there is a condition of somnolence, and the sufferer falls into short, unrefreshing naps, which are disturbed by dreams, and from which he awakes with a start. The skin is not infrequently bedewed by a cold sweat, which about the head and neck may be so copious as to run off in trickling streams.

Stasis within the renal veins leads to scantiness of the urine, which is dark in colour, loaded with urates, and often contains albumin and casts. The action of the bowel becomes irregular and constipated, or as the dropsy invades the abdominal structures the patient may be annoyed by frequent scanty, liquid stools. Congestion of the hæmorrhoidal veins sometimes gives rise to additional distress. Disorders of the pelvic viscera are common at this time in the female; the catamenia are apt to be scanty and irregular, and leucorrhœa is not uncommon. Day by day the distress of the patient increases; during his waking hours he longs for the relief of sleep at night, and by night his discomfort makes him long in turn for the days. Days drag on into weeks, and not infrequently weeks into months, with ever-augmenting dropsy, which at length invades the serous cavities (Fig. 43). Ascites and tumefaction of the abdominal walls intensify pressure upon the diaphragm and abdominal vessels, rendering breathing still more laboured. The pressure thus occasioned still further impedes the return flow from the veins of the lower extremities, and causes an increase of anasarca. If hydrothorax now sets in, the patient's shortness of breath becomes extreme, and he is obliged to support his body by resting his arms on a table in front of him. I have known a sufferer from mitral disease in this stage to remain thus for several weeks, not venturing to leave her chair. Fortunately for these patients, nature is not able long to maintain the unequal struggle, and unless treatment brings relief, death does so ere long.

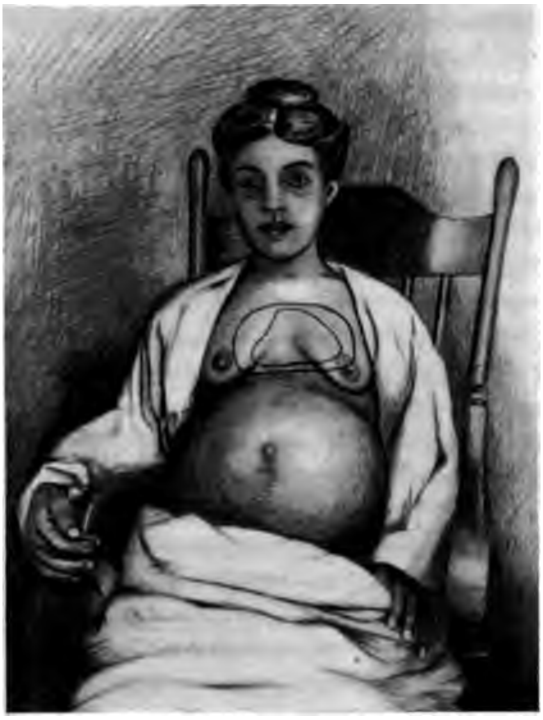


FIG. 43.—CASE OF MITRAL STENOSIS, SHOWING ASCITES AND CLUBBING OF FINGER-TIPS.

Areas of superficial and deep-seated dulness are indicated.

Occasionally in this extreme stage the end comes through sudden stoppage of the heart, but as a rule it is the result of some one of the causes that will be narrated in the part of this subject devoted to the mode of death.

Physical Signs.—*Inspection* is apt to detect more or less cyanosis, and in pronounced cases there may be distinct blueness of the lips and finger-tips. Patients, particularly children, who have had the disease for years usually display clubbing of the terminal phalanges. Often there is bulging of the præcordium, particularly at the lower end of the sternum, as well as visible epigastric pulsation. If compensatory hypertrophy is great, and lung-borders are retracted, the eye may discern a systolic pulsation over the body of the heart and a short diastolic shock in the pulmonary area the same as in regurgitation. The apex-beat is usually feeble, and not likely to be outside of the nipple or below its usual situation.

Palpation confirms the impression received by the eye, but in addition detects a thrill at the apex, which, preceding the ventricular impulse, is known as *presystolic.* This thrill resembles the purring of a cat, and hence is called " fremissement cataire." It may be short and soft, or rough, and extend throughout the greater part of diastole. In some instances a shorter, feebler thrill follows the second sound, occupying the forepart of the diastolic period. The presystolic thrill is found to lead up to, and terminate in a short, sharp systolic shock or " thumping " apex-beat. This thrill is often so short as to convey the impression of the apex-beat being split, the second of the two impulses being the sharper and stronger. A sharp stroke, imparted by the sudden closure of the pulmonic valve, is sometimes felt distinctly in the second left interspace, close to the sternum. Epigastric pulsation is generally pronounced, and gives the impression of a powerfully contracting right ventricle.

In compensated cases of stenosis the pulse is small, feeble, and regular, and less rapid than in mitral regurgitation.

There has been much controversy, chiefly among the English, as to whether the pulse of mitral obstruction or of insufficiency is the more likely to be irregular. This, in my opinion, is a matter of slight practical importance, and yet in my experience I have found the pulse to be more often irregular in regurgitation than in stenosis.

The annexed sphygmographic tracing (Fig. 44) is from a case of pronounced mitral stenosis in a female, and shows the pulse small, of high tension, and regular. When pulse-tension is pronounced, it is due to capillary resistance and not to the energy of left ventricular contraction. Concerning the irregularity of the pulse in mitral disease, it may be again stated that observations of Radizewsky appear to prove that the character of the pulse in this respect depends upon the state of the myocardium of the auricles. When this is healthy, the pulse is regular; when degenerated, either fibroid or fatty, the pulse becomes irregular, even arrhythmic.

Fig. 44.—Sphygmogram from Case of Mitral Stenosis.
(Personal observation.)

Popoff has called attention to the occasional occurrence of a *pulsus differens* in this disease, by which term is meant an inequality in the two radial pulses, the left being the smaller. As this is observed when compensation is destroyed, and may disappear with restoration of cardiac energy, Popoff attributes the inequality to pressure of the greatly dilated left auricle on the left subclavian artery. Preble has also noticed its occurrence in some of his cases. As pulsus differens may also be produced by aneurysm, embolism, thrombosis, arteriosclerosis, etc., it is important that all such causes be excluded before the phenomenon is attributed to extreme dilatation of the auricle, a matter that may be of some moment in prognosis.

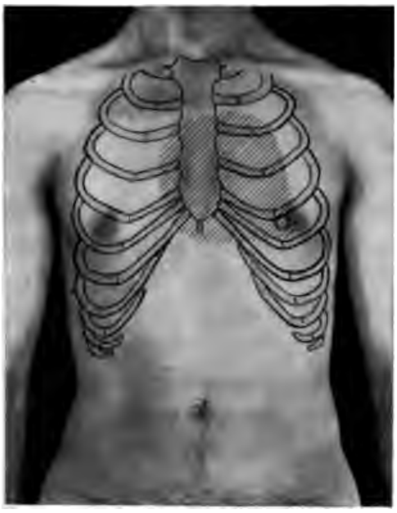


Fig. 45.—Location of Apex-beat and Area of Deep-seated Dulness in Mitral Stenosis.

Percussion shows a similar change in absolute and relative cardiac dulness as described in the article on mitral regurgitation—viz., *an increase of cardiac dulness towards the right side and downward* (Fig. 45). This increase bears a direct relation to the degree of stenosis. According to Leube, percussion shows a more pronounced enlargement of the right heart in this form of mitral disease than in insufficiency, a point he regards as of importance in the differential diagnosis between these two affections.

Another difference lies in the fact that, owing to atrophy instead of hypertrophy of the left ventricle, dulness is not likely to be much if at all increased to the left.

Auscultation.—In pronounced cases of mitral stenosis, auscultation at the apex of the heart detects a murmur of such intensity and distinctive character that it at once fastens the attention of the examiner. In most instances it is a long-drawn, rough bruit, which, beginning after the second sound, runs up to and terminates abruptly in a clear, sharply accented first sound. The murmur is spoken of, therefore, as presystolic, and in this respect corresponds exactly to the thrill already described. When well marked, this presystolic murmur is so striking as to be almost pathognomonic of mitral obstruction (Fig. 46).

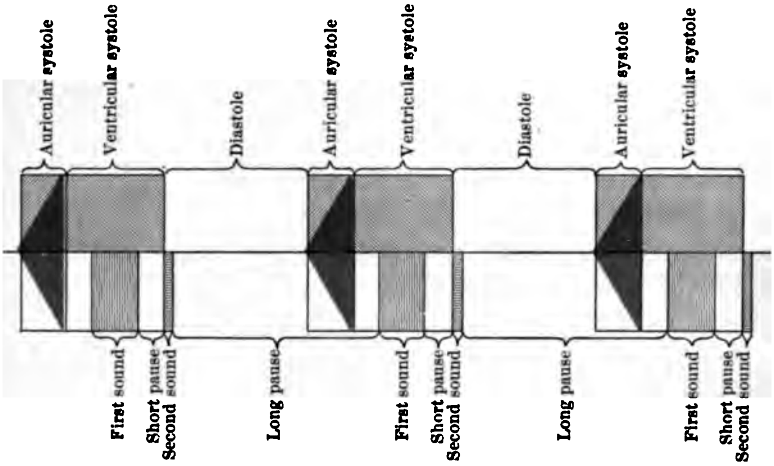


FIG. 46.—RHYTHM OF CHARACTERISTIC MURMUR OF MITRAL STENOSIS, "AURICULAR-SYSTOLIC."

The rhythm of this bruit, by which is meant the time of its occurrence, has been the subject of considerable controversy, for the reason that some observers have declared it to be in reality systolic and only seemingly presystolic. The generation of the first sound, say they, is delayed in consequence of the rigidity of the mitral valve, and hence, although the murmur begins with the contraction of the ventricle, its occurrence prior to the first tone gives it the appearance of preceding ventricular systole. The arguments in support of this opinion have never convinced me of its correctness, and consequently I regard the bruit as truly presystolic.

When we reflect on the physiology of cardiac action we see that a murmur which is audible before ventricular systole is generated during diastole, and that therefore the murmur of mitral stenosis is diastolic. This is not all, however; the bruit in most cases is plainly heard to begin in the latter portion of the long pause—i. e., the diastolic interval—and to end exactly with the first tone. It is synchronous, therefore, with the contraction of the left auricle, which, as we know, takes place immediately before that of the ventricle. For the reason, then, that the murmur is generated during auricular systole, Gairdner long ago proposed the name for it of the "auricular systolic" murmur.

This term is too restricted, however; since, as is well known, the bruit in some cases commences before the contraction of the auricle, in fact immediately after the second sound, and lasts throughout the long pause. It is consequently a diastolic and not always an auricular systolic murmur, and as such is in contrast to the systolic one of mitral regurgitation. It has also been called the "mitral direct" murmur, because transmitted in the direction of the blood-stream—i. e., from the mitral opening directly to the apex of the left ventricle. Indeed, it may be stated *en passant*, that all bruits of stenosis are called direct and those of regurgitation indirect murmurs. Considering, then, the designations that have been given to the murmur in question, the best is the one in most general use, which is presystolic.

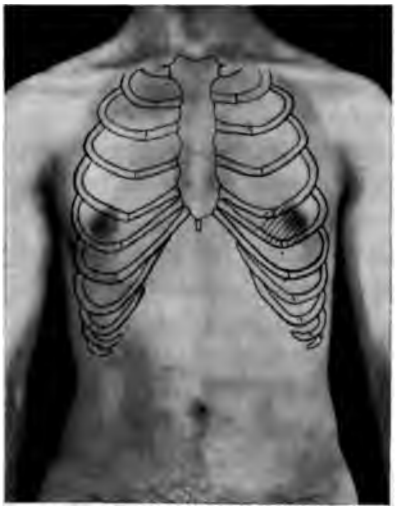


FIG. 47.—AREA OF AUDIBILITY OF THE PRESYSTOLIC MURMUR OF MITRAL STENOSIS. It is frequently limited to this area.

The murmur is heard most distinctly close to the apex-beat, not directly at the seat of impulse, but slightly within and above, at the point, in fact, where the thrill is felt most plainly (Fig.

47). Its area of audibility is sometimes very limited, being confined to the immediate proximity of the apex, but I have known the murmur to be audible for a considerable distance in all directions, although even then it is not transmitted so widely outside of as inside of and above the apex-beat.

The quality or timbre of the bruit is exceedingly rough and harsh, so that it is frequently described as rolling, blubbering, spluttering, etc. Balfour happily describes it in some instances as sounding like V-o-o-t or the sound produced by the attempt to roll out the letters R-r-b, or when still more prolonged R-r-r-b. The final consonant of these combinations is supposed to represent the short, sharp first sound that terminates the bruit. The murmur never possesses the soft, blowing quality of the mitral regurgitant murmur, since *obstructive bruits are always rougher than those of regurgitation.*

The length and intensity of a presystolic murmur are influenced by posture and the rate of cardiac action. Thus a bruit, which is short and rather indistinct when the heart is beating rapidly, or when the patient is standing, is very likely to increase appreciably in duration and to display its true character more distinctly after the individual has lain down and the heart's action has become slower. In other instances the reverse obtains, the bruit being most distinct in the erect posture. One should auscultate in all positions and under varying conditions of cardiac action.

Another peculiarity of the mitral direct murmur is its *changeability,* by which is meant that it is not always the same in distinctness at different times. I recall vividly a woman in whom on several occasions I felt certain of the existence of a mitral presystolic murmur. On one occasion, however, after an absence from observation of several months, her heart presented no such murmur as I had heard before, but instead a feeble first sound accompanied by a faint systolic whiff. Some weeks subsequently, after having taken digitalis and strengthened the contractions of the left auricle, the old-time presystolic murmur reappeared. Broadbent regards this changeability as of great significance in the differential diagnosis between stenosis and regurgitation.

The foregoing description applies to most cases of mitral obstruction, but not to all, and as it is the exceptions that are every now and then encountered, they will now be described. In some instances the bruit is so short that it is scarcely recognisable as separate from and preceding the first sound. I have generally noted in such cases, however, that the first sound is short and thumping, and appears to have prefixed to it a short thrill, which causes the impulse to convey to the hand the impression of its having slidden up to its maximum instead of having given a clean thrust, as does the healthy heart. Difficult as it is to recognise this indistinct or abortive murmur, it is extremely important to be able to do so, since it is in the detection of obscure signs of disease that the skilled physician differs from his unskilled colleague.

The reverse of this short, scarcely recognisable bruit is the long-drawn murmur, which Traube first described and designated the "modified presystolic murmur." This is the murmur which, commencing directly at the close of systole—i. e., immediately after the second sound—extends through the long pause of diastole, and ends with the next first sound (Fig. 48). A not at all infrequent auscultatory finding is a short murmur occurring after the second sound and known as *early diastolic*, and which is then succeeded by a short period of silence, and then a characteristic presystolic murmur (Fig. 49). This anomaly is therefore a breaking in two, as it were, of the long murmur, and by Fraentzel

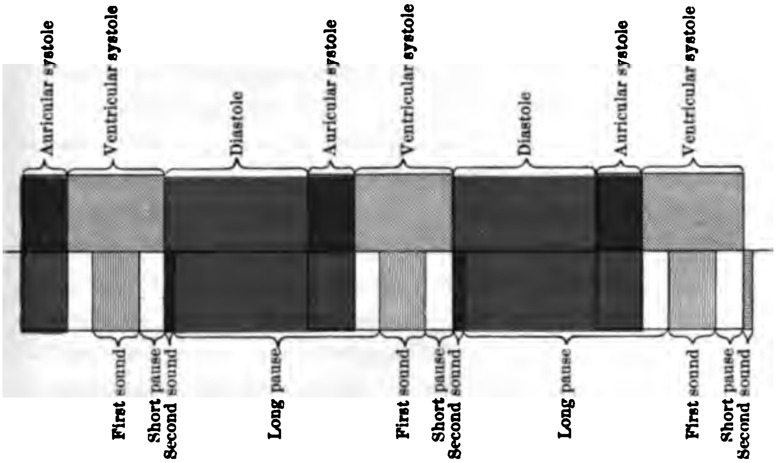


FIG. 48.—RHYTHM OF OCCASIONAL VARIETY OF MITRAL STENOTIC MURMUR, THROUGH ENTIRE VENTRICULAR DIASTOLE.

was called the "*interrupted modified presystolic murmur.*" It is very diagnostic, but may easily mislead an inexperienced auscultator. Should such difficulty of interpretation arise, error may be avoided by due attention to the associated secondary physical signs and to the modifications of the heart-sounds soon to be described.

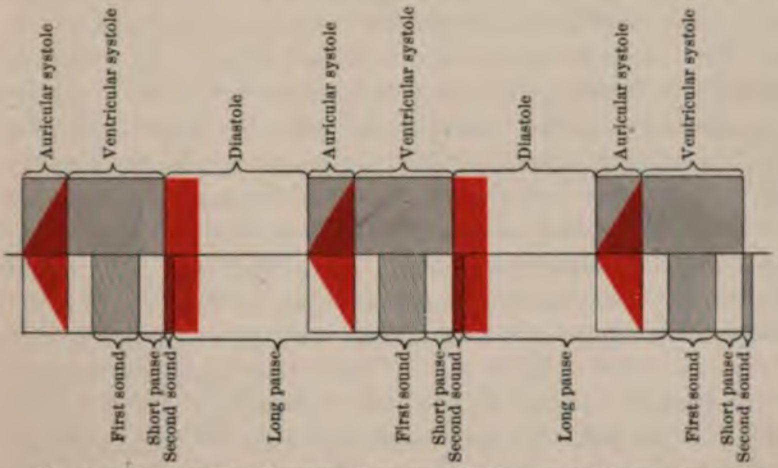


FIG. 49.—"INTERRUPTED MODIFIED PRESYSTOLIC" MURMUR OF MITRAL STENOSIS.

Another departure from what is usually heard in mitral stenosis is the retention of the presystolic bruit and of the first sound *without a second sound*, or of the murmur alone *without either of the cardiac tones.* Attention is directed to these anomalies by Broadbent, who states that under such circumstances it is possible for the murmur to be mistaken for a systolic one followed by a second sound, or for the bruit to be considered systolic, and to have replaced the sound altogether. Care should be taken to avoid such an error, since a systolic murmur means regurgitation, and for sake of prognosis as well as treatment stenosis should be recognised as such whenever it exists. A mistake can probably be avoided by palpation of the carotid pulse, when it will be found that this is preceded by the murmur.

Such comparison of the time of the murmur with that of the carotid pulse is likewise valuable when, as stated by Fraentzel, the presystolic murmur disappears in the last weeks of life, or becomes merged into a systolic one.

The various modifications in rhythm and intensity of the mitral obstructive murmur are due to differences in the rapidity and force with which the blood flows through the narrowed orifice. It is conceivable—e. g., that during the fore part of diastole blood flows too gently into the relaxed ventricle to produce sonorous eddies and vibrations. When, however, it is energetically propelled by auricular contraction, eddies or currents are generated of sufficient force to give rise to the presystolic murmur. In the same manner a diastolic murmur following the second tone owes its production to sonorous eddies generated as the blood gushes out of the auricle into the ventricle. Then as blood-pressure in the ventricle is raised, vibratory, and hence audible currents cease for a time, until auricular systole again throws the blood-stream into sound-producing currents and eddies. Narrowing and roughening of the mitral orifice furnish all the conditions essential for the generation of eddying or whirling currents in the blood-stream as it passes the ostium. Yet if the blood-flow is languid the eddies within it may fail to set up vibrations of sufficient force to be conducted to the ear or hand of the examiner. This explains why shortly before death or during times of great cardiac feebleness the presystolic murmur may disappear, and why it reappears as heart-power is restored.

Heart-sounds.—The recognition of the characteristic murmur of mitral obstruction is not enough; it is necessary to also study and recognise peculiarities in the heart-sounds. In well-marked cases the first tone at the apex is short and valvular, or, as is said, "thumping." This quality is so peculiar and striking as to be quite distinctive and of itself sufficient many times for an experienced auscultator to make a *diagnosis on it alone*. It is the auditory impression of the sharp, quick tap that forms the apex-beat in this disease.

The second sound at the apex may be distinct, but in most cases it is indistinct. At the base of the heart it is sometimes split or reduplicated in consequence of the pulmonic valve being closed a fraction of a second later than the aortic, according to the law that the valve closure is delayed in that artery in which blood-pressure is the higher. In addition also the pulmonic second sound is accentuated.

The phenomenon, however, which is of greatest interest in many ways is what is termed the *simulated or apparent doubling* *of the second sound.* This is to be distinguished from the splitting of the second sound at the base. It is limited strictly to the mitral area, sometimes to the very site of the apex-thrust, and consists in the occurrence of a third tone, which immediately follows the normal second sound. English clinicians have given much study to this apparent doubling of the second sound, and have offered a variety of explanations for its occurrence. The most reasonable theory is, as suggested by Sansom, that it is in some way a sound of valve-tension being produced as the blood gushes forcibly out of the auricle into the ventricle. This seems borne out by the observation that this sound sometimes becomes changed into, or replaced by an early diastolic murmur. Sansom states also that this double sound is heard at some time or other in all cases of mitral stenosis, and indeed may in some instances be the only indication of the lesion.

When this auscultatory phenomenon is present, together with a presystolic murmur, it forms a very striking assemblage of sounds that cannot possibly be mistaken for any other condition than mitral stenosis.

I have known this doubling of the second sound to be inappreciable when the heart was not strong, and to come out clearly and beautifully as treatment restored cardiac power. When the heart beats slowly and regularly it is a matter of no difficulty to differentiate the several sounds and murmurs heard in mitral stenosis. When, on the contrary, the rhythm of the heart is disturbed, the impression may be received of an indistinguishable jumble of sounds, both normal and adventitious. Thus, I have a male patient with a rheumatic mitral narrowing combined with a slight degree of insufficiency who presents such a jumble. When, as now and then happens, his heart's action is tolerably slow and regular I hear the following: A rough presystolic murmur ending in a thumping first sound, then an exceedingly brief pause, followed by a doubled, or apparently doubled, second sound, which in its turn is succeeded by a short, early diastolic murmur and a short silence preceding the next presystolic bruit. At times a short systolic murmur accompanies the first sound, and as this heart is generally very irregular in rhythm it can better be imagined than described what an unintelligible mixture is made by its sounds and murmurs.

Diagnosis.—The diagnosis of mitral stenosis is usually a comparatively simple matter. It may, however, be difficult and next to impossible to say whether it or insufficiency is present. Such a differentiation is important, however, from the standpoint of prognosis and treatment, and should be made when possible. As aids in this direction are the following: (1) Sex, stenosis being more common in females, regurgitation in males. (2) The short, sharp apex-beat preceded by a thrill of longer or shorter duration. (3) The greater extent of dulness over the right heart in stenosis with stronger and more distinct epigastric pulsation. (4) A rougher lower pitched murmur occurring in some portion of the diastole, usually presystolic, but often also early diastolic. (5) Doubling of the second sound, limited strictly to the mitral area or to the apex. (6) The likelihood in stenosis of more pronounced secondary effects in other organs than the heart. (7) The greater smallness and feebleness of the pulse in stenosis, and the greater likelihood of arrhythmia in regurgitation.

As a matter of fact differential diagnosis is not likely to be difficult except in the stage of lost compensation, and then less dependence must be placed on the auscultatory findings than on the evidences of greater secondary effects in stenosis.

In all cases the question of ascertaining the exact nature of the lesion is not all of diagnosis. One has also, or in addition, to decide the degree of the lesion and the severity of its effects, and whether or not the findings account for the symptoms complained of. The degree must be determined by careful consideration of the murmurs, sounds, and secondary effects. The longer the presystolic murmur, the more thumping the first sound and apex-beat, the greater the enlargement of the right and the smaller the left ventricle, the feebler and smaller the pulse, the more pronounced the evidences of secondary effects on the liver—then the more pronounced will be the degree of narrowing. The association of a mitral regurgitant bruit points to a medium degree of stenosis, and so, according to Sansom, does the simulated doubling of the second sound at the apex.

Dyspnœa on even slight exertion, as slow walking, great proneness to cough, and other signs of bronchial congestion, a feeling of weakness and fatigue out of proportion to the effort occasioning it, scantiness of urine, emptiness of the arterial system—are all sympwas called the "*interrupted modified presystolic murmur.*" It is very diagnostic, but may easily mislead an inexperienced auscultator. Should such difficulty of interpretation arise, error may be avoided by due attention to the associated secondary physical signs and to the modifications of the heart-sounds soon to be described.

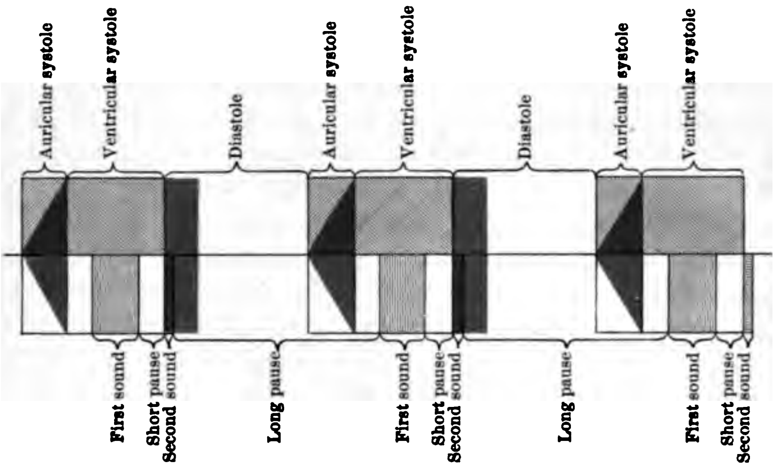


FIG. 49.—"INTERRUPTED MODIFIED PRESYSTOLIC" MURMUR OF MITRAL STENOSIS.

Another departure from what is usually heard in mitral stenosis is the retention of the presystolic bruit and of the first sound *without a second sound,* or of the murmur alone *without either of the cardiac tones.* Attention is directed to these anomalies by Broadbent, who states that under such circumstances it is possible for the murmur to be mistaken for a systolic one followed by a second sound, or for the bruit to be considered systolic, and to have replaced the sound altogether. Care should be taken to avoid such an error, since a systolic murmur means regurgitation, and for sake of prognosis as well as treatment stenosis should be recognised as such whenever it exists. A mistake can probably be avoided by palpation of the carotid pulse, when it will be found that this is preceded by the murmur.

Such comparison of the time of the murmur with that of the carotid pulse is likewise valuable when, as stated by Fraentzel, the presystolic murmur disappears in the last weeks of life, or becomes merged into a systolic one.

The various modifications in rhythm and intensity of the mitral obstructive murmur are due to differences in the rapidity and force with which the blood flows through the narrowed orifice. It is conceivable—e. g., that during the fore part of diastole blood flows too gently into the relaxed ventricle to produce sonorous eddies and vibrations. When, however, it is energetically propelled by auricular contraction, eddies or currents are generated of sufficient force to give rise to the presystolic murmur. In the same manner a diastolic murmur following the second tone owes its production to sonorous eddies generated as the blood gushes out of the auricle into the ventricle. Then as blood-pressure in the ventricle is raised, vibratory, and hence audible currents cease for a time, until auricular systole again throws the blood-stream into sound-producing currents and eddies. Narrowing and roughening of the mitral orifice furnish all the conditions essential for the generation of eddying or whirling currents in the blood-stream as it passes the ostium. Yet if the blood-flow is languid the eddies within it may fail to set up vibrations of sufficient force to be conducted to the ear or hand of the examiner. This explains why shortly before death or during times of great cardiac feebleness the presystolic murmur may disappear, and why it reappears as heart-power is restored.

Heart-sounds.—The recognition of the characteristic murmur of mitral obstruction is not enough; it is necessary to also study and recognise peculiarities in the heart-sounds. In well-marked cases the first tone at the apex is short and valvular, or, as is said, "thumping." This quality is so peculiar and striking as to be quite distinctive and of itself sufficient many times for an experienced auscultator to make a *diagnosis on it alone.* It is the auditory impression of the sharp, quick tap that forms the apex-beat in this disease.

The second sound at the apex may be distinct, but in most cases it is indistinct. At the base of the heart it is sometimes split or reduplicated in consequence of the pulmonic valve being closed a fraction of a second later than the aortic, according to the law that the valve closure is delayed in that artery in which blood-pressure is the higher. In addition also the pulmonic second sound is accentuated.

The phenomenon, however, which is of greatest interest in many ways is what is termed the *simulated or apparent doubling* *of the second sound.* This is to be distinguished from the splitting of the second sound at the base. It is limited strictly to the mitral area, sometimes to the very site of the apex-thrust, and consists in the occurrence of a third tone, which immediately follows the normal second sound. English clinicians have given much study to this apparent doubling of the second sound, and have offered a variety of explanations for its occurrence. The most reasonable theory is, as suggested by Sansom, that it is in some way a sound of valve-tension being produced as the blood gushes forcibly out of the auricle into the ventricle. This seems borne out by the observation that this sound sometimes becomes changed into, or replaced by an early diastolic murmur. Sansom states also that this double sound is heard at some time or other in all cases of mitral stenosis, and indeed may in some instances be the only indication of the lesion.

When this auscultatory phenomenon is present, together with a presystolic murmur, it forms a very striking assemblage of sounds that cannot possibly be mistaken for any other condition than mitral stenosis.

I have known this doubling of the second sound to be inappreciable when the heart was not strong, and to come out clearly and beautifully as treatment restored cardiac power. When the heart beats slowly and regularly it is a matter of no difficulty to differentiate the several sounds and murmurs heard in mitral stenosis. When, on the contrary, the rhythm of the heart is disturbed, the impression may be received of an indistinguishable jumble of sounds, both normal and adventitious. Thus, I have a male patient with a rheumatic mitral narrowing combined with a slight degree of insufficiency who presents such a jumble. When, as now and then happens, his heart's action is tolerably slow and regular I hear the following: A rough presystolic murmur ending in a thumping first sound, then an exceedingly brief pause, followed by a doubled, or apparently doubled, second sound, which in its turn is succeeded by a short, early diastolic murmur and a short silence preceding the next presystolic bruit. At times a short systolic murmur accompanies the first sound, and as this heart is generally very irregular in rhythm it can better be imagined than described what an unintelligible mixture is made by its sounds and murmurs.

Diagnosis.—The diagnosis of mitral stenosis is usually a comparatively simple matter. It may, however, be difficult and next to impossible to say whether it or insufficiency is present. Such a differentiation is important, however, from the standpoint of prognosis and treatment, and should be made when possible. As aids in this direction are the following: (1) Sex, stenosis being more common in females, regurgitation in males. (2) The short, sharp apex-beat preceded by a thrill of longer or shorter duration. (3) The greater extent of dulness over the right heart in stenosis with stronger and more distinct epigastric pulsation. (4) A rougher lower pitched murmur occurring in some portion of the diastole, usually presystolic, but often also early diastolic. (5) Doubling of the second sound, limited strictly to the mitral area or to the apex. (6) The likelihood in stenosis of more pronounced secondary effects in other organs than the heart. (7) The greater smallness and feebleness of the pulse in stenosis, and the greater likelihood of arrhythmia in regurgitation.

As a matter of fact differential diagnosis is not likely to be difficult except in the stage of lost compensation, and then less dependence must be placed on the auscultatory findings than on the evidences of greater secondary effects in stenosis.

In all cases the question of ascertaining the exact nature of the lesion is not all of diagnosis. One has also, or in addition, to decide the degree of the lesion and the severity of its effects, and whether or not the findings account for the symptoms complained of. The degree must be determined by careful consideration of the murmurs, sounds, and secondary effects. The longer the presystolic murmur, the more thumping the first sound and apex-beat, the greater the enlargement of the right and the smaller the left ventricle, the feebler and smaller the pulse, the more pronounced the evidences of secondary effects on the liver—then the more pronounced will be the degree of narrowing. The association of a mitral regurgitant bruit points to a medium degree of stenosis, and so, according to Sansom, does the simulated doubling of the second sound at the apex.

Dyspnœa on even slight exertion, as slow walking, great proneness to cough, and other signs of bronchial congestion, a feeling of weakness and fatigue out of proportion to the effort occasioning it, scantiness of urine, emptiness of the arterial system—are all symptoms indicative of serious circulatory embarrassment, and attributable to the valvular disease.

On the other hand, neuralgic pains in the præcordia and a feeling of fulness or uneasiness in the cardiac region, coldness and numbness of one hand and not the other, or of the hands and not the feet, headache, and prolonged vertigo, the patient being quiet and the pulse not feebler than usual, a feeling of nervousness and restlessness—may all be neurotic manifestations depending on defective nutrition or elimination, and in such cases are apt to be out of proportion to the degree of the lesion and to symptoms distinctive of cardiac disease.

Prognosis.—In general, this is less favourable than that of mitral incompetence, and for two reasons: (1) Obstruction is constant and tends to greater stasis in the pulmonic vessels, in consequence of which the left auricle and right ventricle are subjected to greater strain. They are likely, therefore, to break in their compensation at an earlier period. (2) Mitral stenosis is a *progressive lesion,* and may under the influence of repeated attacks of subacute rheumatism become at length so extreme that life cannot be maintained.

When the narrowing is pronounced there is but a small volume of blood ejected into the arterial system, general nutrition is correspondingly poor, complications on the part of the lungs are more likely, outdoor exercise is difficult if not impossible, normal metabolic processes are interfered with, and general nutrition becomes very defective.

In a word, even when uncomplicated and apparently well compensated, mitral stenosis offers an exceedingly grave prognosis. By some authorities the average length of life is set down as not far from ten years. It stands next to aortic regurgitation in point of gravity.

The following figures are of interest as showing the average age at which death took place in several series of cases. Of Sansom's 61 cases death occurred at 32.7 years. In Hayden's 42 cases death took place at 37.8 years. Of Broadbent's 53 cases it occurred at 33 years for males, and 37 to 38 for females. Samways found that at Guy's Hospital during a period of ten years the average length of life for both sexes was 38.33 years; in less pronounced forms, 43.6 years; more extreme cases, 33.6 years.

palpable, and there was no evidence of free fluid in the abdomen. The urine was scanty, dark-coloured, and contained a trace of albumin. The temperature was 98.6° F. and respirations 28. Her stomach was very irritable, and for several days she had not been able to retain nourishment.

Four hours after her admission her pulse had increased in rapidity and feebleness, and so few of the pulse-waves reached the wrist that the heart-rate had to be counted with the stethoscope. It was beating 180 per minute. Cyanosis had deepened, cough and dyspnœa were very bad, râles had grown more numerous, and the liver had increased in size. Her condition was so critical that she was given a hypodermic injection of $\frac{1}{8}$ of a grain of morphine with $\frac{1}{60}$ of atropine, nitroglycerin $\frac{1}{100}$, and $\frac{1}{30}$ of sulphate of strychnine, this latter to be repeated every two hours during the night. An ounce of sulphate of magnesia was also administered, and a few hours subsequently she was put upon 10-minim doses of tincture of digitalis every four hours. By the next day her condition had improved materially, the pulse coming down to 134, and being rather more regular, but the strychnine, digitalis, and a daily dose of salts were continued. Without detailing all the fluctuations of this patient for the ensuing ten days, it may be said that the pulse showed ever-recurring vagaries, being at one time fairly regular, all waves reaching the wrist, and at others being rapid, irregular, and intermittent. The liver also varied in size, diminishing and increasing according to the persistence and regularity with which the salts were administered. The cough, however, gradually grew less, expectoration diminished, pain left the epigastrium, she retained nourishment, and as a rule got several hours' good sleep each night. As the condition improved, the strychnine was lessened in frequency of administration, but the digitalis was continued. At one time indeed its action was supplemented by strophanthus. At length, by November 30th, her pulse-rate averaged 84, and it was recorded that all the waves reached the wrist. Dulness and râles had left the lungs, but the liver still remained palpable, although smaller in size. The patient then left the hospital abruptly. Two months subsequently she again re-entered, complaining as before of cough and expectoration, but showing no dropsy. Treatment again benefited her, and she again withdrew from observation. I am indebted for the notes of this case to Dr. J. R. Yung, one of the internes at the time.

This case illustrates fairly well the symptoms and amenability to treatment of a case of mitral stenosis in which compensation was broken, but not irreparably so, and in which, with signs of stasis amounting even to a relative incompetence of the tricuspid valve, there was no œdema. In fact, the brunt of the disturbance was borne mainly by the lungs, the dulness and râles, the dyspnœa, cough, and frothy expectoration being the result of the great pulmonary engorgement. It is hard to explain why in such a case œdema is absent, whereas in other individuals with apparently no greater stasis, dropsy will be a marked and distressing feature. It certainly seems to corroborate the view that dropsy depends upon the state of the blood and nutrition of the capillaries, as well as upon the degree of capillary and venous engorgement. This patient subsequently succumbed to a third attack in the hospital.

Mr. B., aged twenty-nine, tailor, consulted me January 9, 1900, on account of great breathlessness upon the slightest effort. He gave a history of rheumatism four years previous, since which time he had suffered with subacute articular pains. Gonorrhœa six years ago, with stricture at present time. With the exception of a "bad eye," nature unknown, at six years of age, has had no other illness. Heart began to trouble him one year after the rheumatic attack, but was not treated for heart-disease until the summer of 1899. His symptoms were great dyspnœa on effort, cough once in a while at morning and evening, vertigo upon exercise, some pain between the shoulders, and poor appetite, but sleep good. His pulse while sitting was weak, small, regular, and 90. The

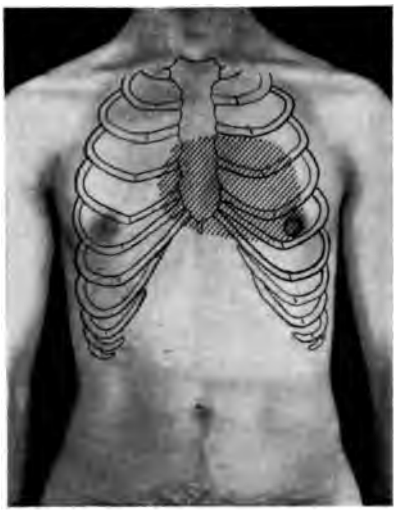


FIG. 51.—LOCATION OF APEX AND RELATIVE DULNESS IN CASE OF MITRAL STENOSIS AND REGURGITATION (p. 273).

examination of the heart discovered the weak apex-beat at the fifth interspace, nipple-line, 3¾ inches from median line, and preceded by a short thrill (Fig. 51). The apex-beat was not thumping, but there was marked epigastric pulsation. Absolute dulness was increased from right border of sternum, at fourth costal cartilage, to left of parasternal line. Relative dulness from lower border of third costal cartilage above to junction of sixth and seventh costal cartilages below, 1¾ inch to right of median line and to ¾ of an inch outside of nipple. The pulmonic second sound was found accentuated. Second sound was not doubled at base, but limited to area of the apex-beat was an apparent doubling of the second sound, the second element at times having the character of a short murmur, and separated from the following presystolic murmur. At lower inner edge of the apex-beat the first sound was also doubled at times.

A short, rough presystolic murmur was found just within, and a blowing systolic at the apex. The murmurs and sounds made a rolling, tumbling rhythm. In dorsal decubitus the apparent doubling of second sound was very marked at inner edge of apex. As the heart occasionally slowed, the first sound was found also doubled, the first element replacing the presystolic murmur. The systolic murmur became plain and whistling with a very pronounced blow 2 inches to left of nipple. The liver was palpable a finger-breadth below the costal arch.

The diagnosis made was mitral stenosis and insufficiency, with secondary cardiac hypertrophy and dilatation.

Mrs. A., aged thirty-three years, weight 115 pounds, height medium, American, was examined March 29, 1901. Her father was living, but had cough, while a maternal uncle and a maternal aunt had died of consumption. At eighteen she had suffered from a severe attack of inflammatory rheumatism, and had had more or less joint pains for three or four years subsequently. Of children's diseases, she had had a mild attack of scarlatina when a child, and thought that during her childhood she had also had pleurisy. She had had a second pleuritis a year prior to her examination by me. Her present illness dated back to 1891, when she first began to have a cough, but her symptoms had grown much worse for the last year, and she had grown perceptibly paler. In the way of symptoms, she complained chiefly of chronic cough, which was most troublesome at night, and of considerable yellowish sputum, in which tubercle bacilli were said to have been discovered. She noticed shortness of breath in walking and ascending stairs. The appetite was poor and the digestion weak, although bowel movements were regular, as also were the menses. Sleep was disturbed by the cough, and there was slight pain in the right hip and the left side of the chest near the shoulder. The voice was husky, but it may be said in passing that laryngoscopic inspection revealed no infiltration of the larynx.

Examination.—The pulse was 105, small, regular, and of noticeably low tension. The temperature taken at 12 M. was 99° F. Respirations were shallow, but not hurried. The chest was moderately emaciated, very shallow in its antero-posterior diameter, and flattened both above and below the right clavicle. Vocal fremitus was increased at both apices, particularly the right. Upon the right side, dulness extended from the apex to the third interspace in front and to the middle of the scapula behind, shading off to impaired resonance as far as the tip of the scapula and below this point, becoming again more pronounced towards the posterior axillary line. In the right infraclavicular region there were bronchial breath-sounds, moist râles of varying size, and the voice-sounds were so concentrated and hollow as to strongly suggest a cavity. Posteriorly, respiratory sounds were also bronchial, and the act of coughing developed numerous fine and coarse crackling râles as far down as the inferior scapula angle. At the left apex there was impaired resonance both front and back to the level of the second rib, and over this area breath-sounds were broncho-vesicular, and cough produced crumpling râles that extended below the limits of slight dulness.

The apex-beat was situated in the fifth left interspace slightly within the nipple-line, was feeble, and of the character of a quick thump, and was preceded by a short yet distinct thrill that ended with the cardiac impulse. Relative heart's dulness was somewhat increased towards the right and downward, but did not reach beyond the vertical nipple-line at the left (Fig. 52). Upon auscultation the first sound at the apex was short, sharp, and thumping, the second sound was not doubled, and the pulmonic second tone was markedly accentuated. A rather short, rough, distinctly presystolic murmur ran up to and ended abruptly with the sharp first sound, and was of greatest intensity in the standing position. A high-pitched systolic whiff accompanied the systole in the mitral area, but was not transmitted to any appreciable distance outside this area.

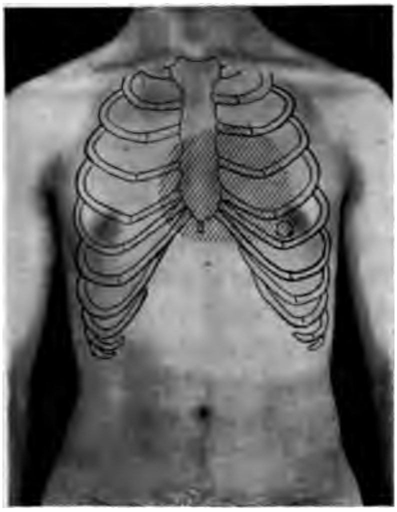


Fig. 52.—Location of Apex and Relative Dulness in Case of Mitral Stenosis (p. 274).

The abdomen was flat and thin, the lower hepatic border distinctly palpable and tender to pressure, and hepatic dulness reached from the upper margin of the sixth rib in the mamillary line to slightly below the inferior costal arch.

The diagnosis was clearly chronic pulmonary tuberculosis with softening and vomica in the right lung, incipient disease of the left upper lobe, mitral stenosis of first degree, with probably some regurgitation, the valvular lesion being of rheumatic origin and in good compensation.

This case is interesting because of the rather rare association of pulmonary tuberculosis and mitral stenosis, and would seem to corroborate the view that this valvular lesion is sometimes of tuberculous origin, were it not for the very definite history of inflammatory rheumatism at the age of eighteen. It likewise shows the fallacy of Rokitansky's statements concerning the antagonism between mitral stenosis and consumption. It is worthy of note, however, that in this case the narrowing was not extreme and was combined with regurgitation, a form of mitral disease which is not so infrequently associated with pulmonary tuberculosis as was once thought.

Furthermore, this case raises the very interesting query if this stenosis may not have exerted a retarding influence upon the progress of the lung affection, although in this connection it should be stated that her residence has been in southwestern Kansas, where the air is dry and the altitude not far from 2,000 feet.

For my part I am much more inclined to attribute the slow advance of the pulmonary affection in this case to other factors, possibly to climatic influences, possibly to inherent mildness of the tuberculous infection itself, rather than to the mitral obstruction, since, as suggested by Sansom, it is reasonable to assume that a valvular lesion would have a tendency to impair the resistance of the organism, particularly in one inheriting a predisposition to tuberculous disease.

CHAPTER VIII

AORTIC REGURGITATION

In this form of valvular disease a portion of the blood discharged into the aorta with each ventricular systole leaks back into the left ventricle during its diastole. Although relative incompetence may be produced by dilatation of the aortic ostium, the disease in question is in the vast majority of cases due to structural defect of the valve itself.

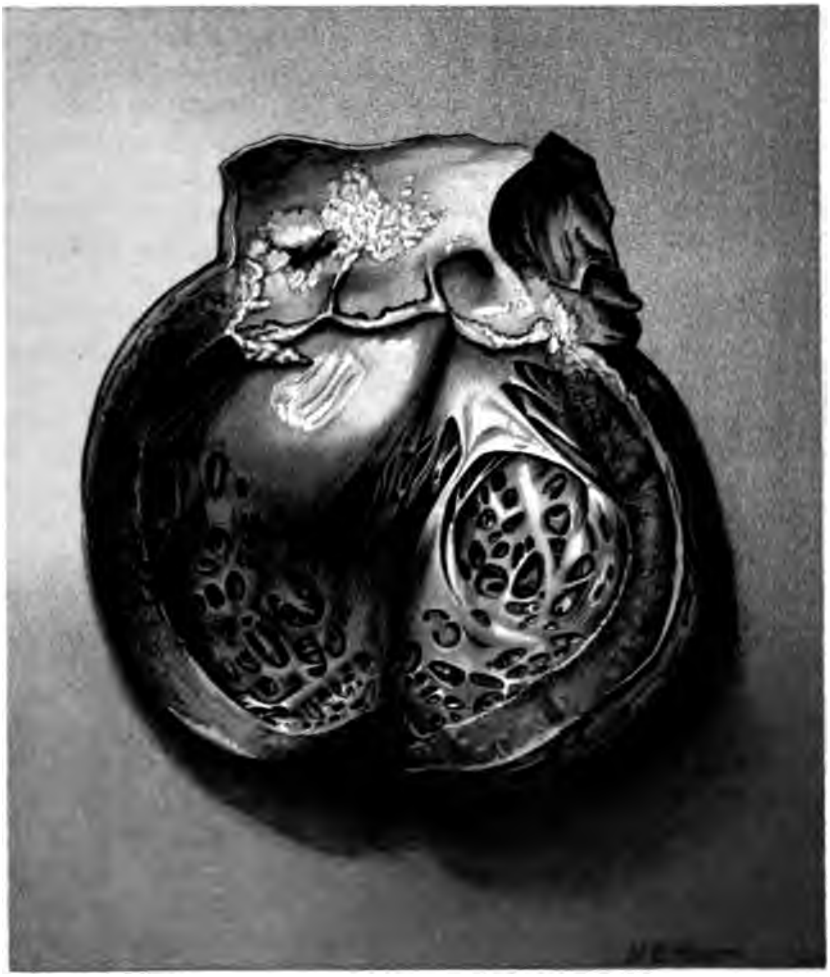
Morbid Anatomy.—Defects in the aortic valve leading to regurgitation are as nearly analogous to those found in mitral regurgitation as the anatomy of the valve will allow. They may follow acute endocarditis, or may be the result of a non-inflammatory sclerosis. The latter is more often the case here than at the mitral orifice because the aortic valve has to bear the brunt of the increased blood-pressure due to muscular exertion.

The leaflets, one or all, may be retracted, curled, or shrivelled, so as to permit a free regurgitation. Old vegetations on the ventricular surface may interfere with their complete apposition. In short, the conditions parallel those found in mitral insufficiency, with exception of the influence of contraction of the chordæ tendineæ and papillary muscles.

Acute incompetence may occur during ulcerative endocarditis by the perforation of one of the valve-cusps. Very rarely a cusp may rupture during violent muscular exercise. The aortic semilunar valve is one of the most delicate structures in the body, and yet one of the strongest, sustaining as it does the whole blood-pressure of the systemic circulation. It is hence extremely unlikely that muscular exertion could be severe enough to raise blood-pressure to a height sufficient to rupture a healthy valve. Probably in such cases the valve has been weakened either by degeneration or inflammation.

As in mitral disease, regurgitation is often combined with

PLATE II



AORTIC REGURGITATION, WITH CALCIFIED VEGETATION THAT SWUNG IN BLOOD CURRENT, CAUSING ATHEROMA OF ENDOCARDIUM AND OF INTIMA OF AORTA.

some degree of stenosis, but regurgitation may occur without narrowing, and occasionally, in consequence of dilatation of the ventricle, even with stretching, of the aortic ring. That such enlargement of the ring, leading to relative insufficiency, could take place, was long doubted, owing to the great strength of the annulus fibrosus, but so many instances of the kind have been observed that there is no longer any room for doubt.

The first effect on the heart is dilatation of the left ventricle. This is due to the impact on its inner surface of the regurgitant stream, which in very free regurgitation re-enters with nearly the force with which it was driven out of the ventricle. Such lesions, however, are of gradual development, and the increasing work leads to a corresponding hypertrophy of the wall of the ventricle, which enables it not only to withstand the strain of the regurgitation, but to expel the greatly increased volume of blood present in the chamber at the beginning of its systole. This hypertrophy in aortic insufficiency is of early development and often becomes so extreme that some of the largest hearts on record are those showing this defect. The wall of the hypertrophied ventricle may be as thick as 4 centimetres (1⅗ inch). The apex of the left ventricle projects far beyond that of its fellow, and the interventricular septum is displaced, encroaching largely on the cavity of the right chamber.

As long as the mitral valve remains intact the effects of aortic regurgitation upon the heart are limited to the left ventricle. If, however, the mitral is incompetent, either from disease of the valve or relatively from the enlargement of the ventricle, the phenomena described as the results of mitral incompetency are added to those of the aortic lesion. In such an event, of course, the right heart is also enlarged, and the largest hearts have been those showing this combination of lesions. Such a heart may weigh as much as 3 or 4 pounds. Indeed, von Ziemssen has reported 6 pounds as the weight of a specimen obtained from one of the great Stokes's patients. On account of its size such a heart is spoken of as *cor bovinum*. The heart presented to me by Dr. C. C. O'Byrne weighs 2¼ pounds, and in it the regurgitation could not have been extreme (Plate II). The point of interest in this specimen is the swinging vegetation, 3 centimetres long, and containing calcareous nodules, which evidently swung in the blood-stream, now in the ventricle, and again in the aorta, for on the intima of the aorta and on the mural endocardium of the ventricle, at the points where the vegetation must have struck, are marked atheromatous patches. There is said to have been a musical murmur during life.

The greatly increased force with which such a ventricle propels the blood into the aorta throws great strain on the walls of that vessel, and hence atheromatous changes are often found not only in the aorta, but in the whole arterial system. That such change is due to this valvular disease is indicated by the fact of its occurrence in young and otherwise healthy individuals, in whom it would not be expected to exist.

When this valvular disease is the result of a general sclerosis, the myocardium is apt to be so degenerated as a result of coronary involvement that hypertrophy is not great. It is when the disease develops in young and healthy individuals as a result of endocarditis that the enormous heart is usually found.

Etiology.—Endocarditis affecting the semilunar valve may have the same origin as that of the left auriculo-ventricular valve, and hence the discussion of its causes does not need to be repeated.

Aortic regurgitation may be met with in persons of both sexes and of all ages. It is a striking fact, however, that this lesion, even when due to rheumatic endocarditis, is far more common in males than in females. When developed at or after middle age, it is usually due to those conditions which bring about sclerosis, and which are fully considered in other chapters (pages 201 and 741). This sclerotic form is also undoubtedly met with more frequently in males than in females, and for the reason that arterial degeneration is more common in the former—and therefore the etiological factors leading to sclerotic change in the aortic cusps are essentially those of arteriosclerosis.

In a considerable portion of males suffering from aortic regurgitation there is a history of syphilis and the abuse of alcohol. In a most typical case of this lesion recently seen in a man of thirty-nine, syphilis and whisky were the only two causative factors to be elicited. Gout and bodily toil, particularly if combined with alcoholic excess, also seem to be causative agents of considerable importance.

As already stated incidentally in Morbid Anatomy, severe strain may bring about acute incompetence of one of the aortic cusps through rupture at some point that had been previously weakened by inflammation or atheromatous degeneration.

There is a form of aortic insufficiency which, although not due to valvular defect, yet presents the same clinical features as the organic form, and is so frequently encountered that it may here be briefly dwelt upon. This is a relative incompetence of the semilunar valve, and its causes are found in conditions that predispose to stretching of the ventricular wall and of the basal ring of the aorta. They are therefore (1) degenerative changes in the myocardium, (2) diseases of the aorta that greatly narrow its lumen, or, *per contra,* lead to its dilatation, and (3) mediastinal tumours, which by pressure diminish the calibre of the aorta. The most frequent cause of this relative insufficiency is aneurysm affecting the ascending arch, or a general dilatation of the aorta secondary to sclerosis. In one instance of the latter kind coming under my notice it was associated with mitral regurgitation, also of atheromatous type, but which had existed for years. During the later weeks of life in this case aortic incompetence developed, and after death was found due to extensive atheromatous degeneration and dilatation of the aorta, reaching from its origin to the beginning of the descending portion of the vessel.

In another case in which regurgitation through the aortic valve had run its course to a fatal termination within a few months, post-mortem examination disclosed stenosis of the ascending aorta, about ½ an inch above the insertion of the valve, so pronounced that the lumen was diminished by at least a half. This narrowing was caused by a growth of fibrous tissue which completely encircled the aorta, and from the history appeared to have originated in acute inflammation a year and a half previously.

As already stated, relative aortic incompetence may be the ultimate effect of chronic myocarditis, which is associated with sclerosis of the aorta, and which so seriously impairs the resisting power of the ring that it gradually yields to the distending force of the blood-wave as it recoils against the closed valve. I have seen more than one instance of the kind as disclosed by the necropsy, although during life the regurgitation had been attributed to structural defect of the valve-segments.

Of 53 cases of aortic regurgitation of which I have records, 46 occurred in the male and only 7 in the female sex. Seventeen of the males and 4 of the females were below the age of forty. Eleven of the former who were less than forty years of age gave a history of rheumatism or scarlatina, while 10 over forty also had had one or the other of these diseases. Of the females, 2 below forty and 1 over that age, gave a history of rheumatism or scarlatina. Of the total number of cases, therefore, 24 were probably due to endocarditis. It may also be stated that of the 29 males and 3 females over forty there were 19 males and 2 females in whom the lesion, owing to the absence of probable endocarditis, could be reasonably attributed to atheroma. In whatever way these figures are looked at they exhibit the striking preponderance of men over women afflicted with this particular valve-defect.

Symptoms.—It goes without saying that in this as in other valvular diseases it is the degree of compensation which determines the presence or absence of distinctively cardiac symptoms. In other words, if the lesion is of inflammatory origin, and if the state of the myocardium has permitted the development of great hypertrophy, the disease may remain entirely latent for many years. Arduous occupations requiring great physical effort, feats of endurance and skill, mountain-climbing, running, boat-racing, football playing, tennis, etc., are often endured without discomfort. I recall an attorney with pronounced aortic insufficiency of rheumatic origin who consulted me soon after his return from a six-weeks' vacation in Colorado. He had ridden his wheel at an altitude of 6,000 feet with no more discomfort than he would have experienced in Chicago. The only time he had suffered any inconvenience was when he had taken the train up Pike's Peak. Upon reaching the summit, 13,000 feet, he fainted away, yet upon returning to the foot of the mountain he got on his wheel and rode away as if nothing had happened.

Dyspnœa is not experienced in this stage, and aside from violent action of the heart, patients are totally unconscious that the organ is anywise different from that of their fellows. If any disturbance of bodily function exists, it is not such as arises from venous congestion, for so long as the mitral valve remains competent stasis back of the left ventricle is impossible.

The state of the circulation is one of *intermittent anæmia* on the part of the arterial system. At each systole the arteries are flushed, and with each diastole they are relatively depleted. The tendency, therefore, is to a lack of nutrition of the various organs and tissues throughout the body. This is not specially manifest in some persons, while in others there is more or less pallor and delicacy of body. The muscular system in particular is weak, and some children show inability for sustained mental effort, yet as a rule young persons with aortic regurgitation show nothing either in appearance or deportment to indicate the existence of their lesion.

A well-compensated aortic insufficiency is not likely to incapacitate an otherwise healthy young adult for the active and even the arduous duties of professional or mercantile pursuits. Many a hard-worked medical man with this lesion is able to sustain the severe mental and physical strain of a large general practice without more fatigue than his more fortunate *confrères*. The only symptoms experienced by some patients are palpitation or slight vertigo, or both, and yet trivial as they may seem to be they are sometimes the earliest announcement of faltering energy on the part of the left ventricle. In one instance, more than twenty years before the patient's death, any effort or excitement beyond a certain moderate degree, brought on attacks of such violent palpitation as to necessitate absolute repose for hours in the recumbent posture. These attacks were also produced by even small doses of digitalis, and as they were allayed by aconite they were thought due to extreme hypertrophy. In most cases such violent cardiac action is an expression of weakness rather than of excessive strength, as sometimes supposed. When dizziness is experienced, it is usually, though by no means always, induced by sudden exertion, and in such cases it is generally found that the regurgitation is very free.

The cerebral arteries are flushed with each systole, but in consequence of the regurgitation blood-pressure within them is not sustained, and when for any reason the reflux into the ventricle is intensified, transient anæmia of the brain results and vertigo is felt. In some cases dizziness is produced by intermittence in the heart's contractions, and it is then a mild manifestation of what in other cases becomes a syncopal attack. Indeed, fainting is an occasional symptom in this disease, in consequence of the fact that the state of the cerebral circulation is the opposite of what obtains in mitral stenosis. This explains, I think, why it is that aortic patients are able to lie low in bed, whereas those suffering from mitral disease usually prefer to sleep with their head and shoulders propped up on two pillows. In the former there is an unconscious attempt to overcome the force of gravitation upon the cerebral circulation, while the latter class of patients seek to aid venous flow out of the head by that same force of gravitation.

Disorders of digestion are not so common in aortic as in mitral patients, and when present are referable not to interference with the circulation, as has been explained is the case in lesions at the auriculo-ventricular orifice, but they are due in most instances to errors in diet or whatever deranges gastro-intestinal function in individuals who have no heart-disease.

The comparative immunity from symptoms enjoyed for years, it may be, by persons whose disease is the result of endocarditis, is not the fortunate lot of those in whom the aortic valves have become incompetent in consequence of sclerosis, and in whom the myocardium is incapable of maintaining adequate compensatory hypertrophy. In persons, therefore, whose signs of aortic regurgitation develop during middle age, symptoms are apt to appear early and to be pronounced. These do not differ essentially from those experienced by patients of the other class, and are palpitation, vertigo of more or less intensity and frequency, a feeling of general weakness, uncomfortable præcordial oppression, more or less pain that may be distinctly anginal or of an anginoid character, and in particular distressing attacks of dyspnœa. When these symptoms arise a fatal termination is not far distant.

With Dr. G. W. Webster, October 13, 1900, I saw an Irishman who presented a very striking picture of the distress often experienced in the terminal stage of aortic incompetence. There was no history of inflammatory rheumatism, but of syphilis and the immoderate use of alcohol. Three weeks prior to my visit he had been in the country, and while there had overexerted himself, become greatly fatigued, and had begun to suffer from attacks of sudden weakness with a feeling of suffocation. Upon his admission to the hospital he was suffering from frequent attacks that seemed to portend speedy dissolution. His hurried and somewhat laboured respirations would suddenly become so augmented in severity that he would spring into the upright position gasping for breath, coughing and raising frothy mucus, while cyanosis became marked, and the pulse grew rather more rapid, irregular, and extremely feeble. The face became anxious, and, in a word, the whole appearance of the man was one of direst distress. The examination on admission disclosed aortic regurgitation and a secondarily leaking mitral, with very swollen and tender liver. Hypodermics of morphine relieved these attacks in a measure, and under the free use of cathartics, digitalis and iodide of soda, the condition so much improved that the mitral no longer leaked.

I found a rather spare man of medium stature who looked much older than he really was. He was semi-recumbent, and although comfortable was yet breathing with apparent difficulty and greater than normal rapidity. There was no œdema, and signs of stasis were not noticeable. The temporal and cervical arteries throbbed strongly, the pulse was quick and of the character known as bisferiens, and the radial arteries were stiff. The lower border of the liver was palpable a short distance below the costal arch, but the organ was not hard or tender. Cardiac impulse was diffused and weak, the indistinct broad apex-beat being in the sixth interspace, midway between the left mamillary and anterior axillary lines. Percussion showed slight increase of cardiac dulness to the right of the sternum and downward, but very great extension of dulness towards the left and downward. The outer border was broadly rounded, after the manner often described as indicative of prepondering dilatation of the left ventricle in cases of aortic insufficiency with broken compensation (Fig. 53). When hypertrophy predominates, the outline of the

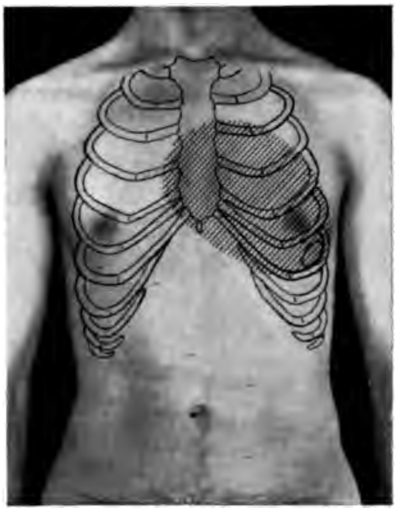


FIG. 53.—LOCATION OF APEX AND RELATIVE DULNESS, CASE OF AORTIC INSUFFICIENCY (p. 284).

left ventricle is long and pointed with a rather sharp apex. Upon auscultation there was at once detected a systolic and diastolic murmur of the usual character in aortic regurgitation, but with the exception of the accentuated pulmonic second tone the heart-sounds were scarcely audible. Indeed, the aortic second was quite wanting, and in the neck was replaced by the feeble distant diastolic bruit. At the seat of the apex the ear perceived a dull or toneless thud rather than the normal first sound, and the diastolic murmur was faintly distinguishable. In the femoral artery, pressure elicited the double murmur of Duroziez.

The diagnosis was easy enough. It was an aortic regurgitation of atheromatous origin in the stage of ruptured compensation that had led to venous stasis. The congestion of the lungs was shown by the frequent cough and sero-mucous sputum, and the stasis in the general system by the hepatic enlargement and scanty urine. The patient was unable to sleep, and the taking of food was followed by the formation of gas, which contributed its quota to the already existing dyspnœa.

The prognosis was of the worst, for it was only too evident that the left ventricle could not withstand the impact of the regurgitating stream. That this was free was shown by the absence of the aortic second sound, which had become wholly replaced by the diastolic murmur. The attacks of increased dyspnœa and cardiac feebleness were the manifestation of left ventricle failure or asystolism and portended grave danger. In fact, although as vigorous and skilful treatment was maintained as could be devised, it exercised no appreciable effect on the patient's condition, and after lingering another five days he expired in one of his attacks.

There is a twofold reason why aortic regurgitation of the sclerotic type is particularly serious. Not only has more or less myocardial degeneration preceded the development of the valvular defect, but the reflux of a portion of the contents of the aorta into the left ventricle augments the cardiac ischæmia resulting from the aortic and often coexisting coronary sclerosis. I will not discuss the much-debated question whether the coronary arteries are flushed during systole or diastole, since this is amply set forth in works on physiology, but only state that the weight of evidence is in favour of the systolic flushing of the heart-muscle.

It is sufficient to emphasize the fact that in aortic regurgitation blood-pressure is not sustained within the coronary any more than in other arteries, and hence cardiac nutrition cannot be good.

There comes at length a limit in all cases to cardiac hypertrophy because the heart-muscle becomes more or less degenerated, and therefore incapable of maintaining the circulation and of withstanding the dilating force of the regurgitant stream. Its flagging energy is shown by more rapid and perhaps less regular contractions, even, it may be, by occasional intermissions.

Therefore, the earliest and most reliable indications of failing compensation are generally shown in the pulse. Even before subjective symptoms bring the patient to his medical adviser the pulse-waves are no longer of uniform frequency, force, and volume. The radial pulse is accelerated, but it does not strike the palpating finger with its old-timed suddenness and vigour, the artery not being so powerfully and quickly distended as when the ventricle contracts with energy. Consequently the physician may not so readily distinguish the peculiar characters of the aortic regurgitant pulse. At irregular intervals the pulse seems to falter a little, or a small, weak beat follows its predecessor more quickly than usual, and is followed by others of normal strength.

This is the expression of an accessory or extra systole, introduced now and then into the regular series of contractions for the purpose of re-enforcement (pulsus intercurrens), or it is the result of the ventricle giving a hurried, incomplete contraction, in consequence of fatigue. As muscular incompetence increases, the pulse grows more irregular, or indeed becomes permanently intermittent. It increases in frequency, and its distinctive collapsing character, to be subsequently described, grows less apparent.

Subjective symptoms annoy or even alarm the patient, who begins to notice an unwonted breathlessness. Attacks of vertigo or even syncope supervene. If the patient does not now die suddenly and unexpectedly, he is likely to suffer from irregularly recurring attacks that are of grave danger because indicating imminent cardiac paralysis. These are a more or less sudden feeling of great weakness or prostration, with cyanosis, a feeble, irregular, perhaps accelerated and empty pulse, dyspnœa, and an indescribable feeling of impending dissolution. In addition, he may suffer from cough with frothy, it may be bloody, expectoration and other symptoms indicative of stasis in the pulmonary vessels and general venous system. If the mitral valve has become relatively incompetent, regurgitation through the auriculo-ventricular opening is added to that already present at the aortic orifice, and the symptoms become the same as those of the last stages of mitral disease.

Early in my practice I was called to attend a middle-aged woman, whom I found intensely dropsical, orthopnœic, and presenting unmistakable evidence of aortic and relative mitral insufficiency. Rest in bed, infusion of digitalis and catharsis speedily removed the anasarca, closed up the mitral valves, and, in short, so greatly improved her condition that she thought herself fully restored. Despite my warning, and contrary to my strict orders, she insisted upon leaving her bed and sitting dressed in the family living room. Only a day or two thereafter, while alone in her apartment, she heard a rap on the door, arose quickly to answer the knock, opened the door, and almost immediately fell to the floor and died. In this case I believe life might have been prolonged had the mitral valves continued to leak, and thus acted as a safety-valve for the left ventricle.

I am led to this opinion by my observation of a case with Dr. Lorence at Chebanse, Ill. The patient was an old man, who was suffering from albuminuria, dropsy, congested liver, and orthopnœa. Upon examination, there were the usual signs of stiffened arteries and aortic regurgitation, but, in addition, a mitral regurgitant murmur, pulsation of the external jugulars, and a murmur characteristic of tricuspid insufficiency. The outlook seemed very bad, and but small hope for improvement was held out. Nevertheless, upon the daily moderate use of cathartics and nitroglycerin, the replacement of digitalis by strophanthus, and the hypodermic administration of small tonic doses of morphine, this patient actually improved beyond all expectation, and several months subsequently was reported by the doctor as still alive and in tolerable comfort, being able to drive out in pleasant weather, although, needless to say, compensation was never restored. I believe in this instance the leakage of the mitral and tricuspid valves relieved the two ventricles from dangerous strain and threw the brunt of the trouble back upon the liver and general venous system. The stasis thus resulting, of course, produced respiratory embarrassment and functional derangement of the abdominal viscera, but actually served to prolong life.

In my care in the wards of Cook County Hospital there are at the present writing two men with aortic regurgitation in whom mitral incompetence has become added. Both present evidence of venous stasis in a moderate degree, chiefly hepatic and pulmonary. One complains of weakness and but little else, the other of insomnia; yet in both patients things are growing slowly worse in spite of rest in bed and the usual heart-tonics. To all intents and purposes they have become converted into cases of mitral disease, the most frequent sequel of events in aortic regurgitation. The chief difference, however, lies in the refractoriness to treatment and in the liability, one might almost say certainty, of a sudden death.

In June, 1899, I saw with Dr. Houston a powerfully built Irishman, weighing over 200 pounds, who was suffering from dyspnœa, which had suddenly developed six weeks previously. His personal history was negative with exception of swelling of one knee some six or eight years before. This may have been a monarticular rheumatism, and if so, it may have been responsible for the man's valvular disease. It was not followed by any symptoms, for with exception of a fall that occasioned pain near the heart for a day he had been perfectly well up to his present illness. No history of overexertion or any other exciting cause for his dyspnœa could be elicited. His shortness of breath had set in abruptly while he was attending his duties as engineer, and at first had been more severe than it was when I saw him, the improvement being due to treatment. Nevertheless he was incapacitated for work, and counsel was sought in the hope of obtaining some suggestion for his further improvement.

The pulse was arrhythmic and rapid, displaying feebly the usual characters of aortic insufficiency, and the vessels did not feel thickened and stiff. The broad, heaving apex-impulse was displaced downward into the sixth interspace and outward to the anterior axillary line. There was epigastric pulsation and a systolic thrill in the aortic area. The heart-tones were everywhere audible, though feebly, and there was a loud, rough systolic murmur at the base to right of sternum, followed by a very feeble diastolic bruit. At the apex could be made out a softer systolic murmur possessing the characters of a mitral regurgitant one, which, together with evidences of enlargement of the right heart, convinced me that the mitral as well as the aortic valves were leaking. The liver was palpable and tender, and the urine contained a small amout of albumin. I looked upon the mitral insufficiency as relative and secondary to the aortic disease.

The prognosis was very unfavourable, notwithstanding the degree of improvement that had already attended treatment, because when compensation is once lost in this form of valvular disease it is rarely possible to restore the dilated and perhaps degenerated left ventricle to its former vigour.

The patient was informed of his grave state, and was advised to keep his room for as long a time as was thought best, the duration to be determined by results. In addition to rest, the treatment was to consist of strychnine, digitalis, and nitroglycerin; food was to be light but sustaining, and cathartics were to be employed daily, but not enough to weaken him. The purpose of the last-named remedies was chiefly to prevent the patient from being obliged to strain at stool, as might be the case were he to become at all constipated. It is well known that effort of this kind is particularly bad, even dangerous, for persons whose left ventricle is in a state of dilatation. In aortic regurgitation the sudden constriction of the arteries incident to straining is liable to cause sudden and fatal diastolic arrest of the heart.

In October I saw the patient again, and was not surprised, although disappointed, to find that in spite of treatment the dilatation of the left ventricle and resulting insufficiency of the mitral valves had increased. The action of the heart was more regular, but in other respects things had grown rather more ominous. He was now ordered to keep his bed strictly, and was put on larger doses of digitalis. Improvement did not follow, and he began to suffer much from sudden paroxysms of dyspnœa, which were very alarming to him and his friends. His liver also became greatly engorged, and his whole condition grew steadily more threatening. Strychnine and nitroglycerin were increased, and he was given daily injections of morphine, ⅛ of a grain, with atropine to lessen dypsnœa, quiet his nervousness, and sustain his heart.

It was decided to persevere in the use of digitalis, interrupt-ing it from time to time and substituting therefor tincture of strophanthus, to prevent the possible cumulative action of the foxglove. This was carried out until at length a singular mental state developed, characterized by delusions closely resembling a mild mania. As Dr. Houston had observed a similar mental state once before in a patient whom I had turned over to his charge, and in that instance had discovered it was caused by digitalis, he concluded it was of the same nature and origin in this case and promptly stopped the drug. As in the other case, so also in this, the delusions and other maniacal manifestations lasted about twenty-four hours, and then disappeared entirely. This rare effect of the prolonged administration of this agent will be spoken of in the chapter on Treatment of Valvular Diseases.

Patient was seen again in January. He was still in bed, where he had remained since the fore part of October, was quite recumbent, and breathing tranquilly, although he stated he had occasional paroxysms of dyspnœa that compelled him to spring up for breath. These spells of difficult breathing had returned upon him about the 1st of January after a period of constipation. His physician stated that as a result of vigorous purgation, digitalis, strychnine, and morphine hypodermically, and restricted diet, which was kept up for nearly two months, his condition had by late autumn improved wonderfully. The enlarged liver had returned nearly to normal, his colour had grown quite natural, and his pulse stronger, of better volume, and regular, the heart-sounds stronger, and the apex-beat fairly well defined. Recently, however, the patient had become intolerant of the cathartics and prolonged rest in bed, and had implored to be allowed to sit up.

I found the pulse about 70, with two intermissions in a minute and a half, but very compressible, and its collapsing character not well marked. The liver was palpable, particularly the left lobe, which was very tender in the epigastrium. There was no œdema, although the feet were a little puffy. Cardiac impulse was wanting except for an occasional vague apex-beat considerably outside the left nipple in the sixth interspace. Heart's dulness was pronounced, presenting in this regard a marked contrast to its condition in October, when it was obscured by pulmonary resonance. It was of triangular outline, reaching to the third interspace, and from 2 inches to right of sternum across nearly to the left anterior axillary line, well outside of the occasionally palpable impulse. The lungs were everywhere resonant. The heart-sounds were audible though faint, the aortic second being particularly feeble, and the pulmonic second accentuated. A loud, harsh murmur was present throughout the præcordium, which was systolic, but could not be traced to any particular area. Over the body of the organ it disappeared on firm pressure, permitting other more distant and persistent murmurs to be distinguished. These were found to be a harsh aortic systolic, a soft mitral systolic transmitted to the back, and a feeble diastolic, which was of aortic origin, as shown by its area of intensity and direction of propagation.

The diagnosis was apparent; added to his old-standing aortic insufficiency with relative mitral regurgitation there was a pericarditis with moderate effusion. The liver was both congested and displaced downward.

The prognosis was most unfavourable, for in addition to the cardiac dilatation depending largely on myocardial degeneration, a pericarditic effusion had taken place. As is well known, this sometimes supervenes upon a chronic valvular disease, particularly aortic insufficiency, and is then apt to be a terminal event. It was stated to the family that sudden death was not improbable.

In the way of treatment, compound cathartic pills were ordered, the patient objecting to elaterium and disagreeable saline waters; digitalis in considerable doses, a grain of codeine thrice daily to promote quiet and to lessen the paroxysms of dyspnœa, and restricted diet. Morphine was not prescribed because of its constipating and other objectionable effects.

In spite of the greatest possible care this patient did not improve, and one month later died suddenly and quietly while resting in bed, as usual. This case not only portrays the clinical picture often seen in aortic regurgitation, but also illustrates the powerlessness of our art in attempting to stay the progress of the disease.

In this and Dr. Webster's case there was one symptom common to both—i. e., paroxysmal dyspnœa. In the one case it seemed due to sudden threatening asystolism of the left ventricle, as shown by feebleness, rapidity, and irregularity of the pulse, and pulmonary congestion, manifested by cough and frothy expectoration. In the other there was also threatening weakness of the heart's action, but the striking concomitant was the intense anxiety amounting to fear, so that the patient would spring up in bed gasping for breath and looking wild and terrified. In both these instances, moreover, the symptoms of cardiac breakdown developed suddenly, and were never again wholly lost, in this respect differing markedly from the gradual onset of compensatory failure seen in mitral affections. In both cases mitral regurgitation was superadded, but instead of the end coming with pronounced dropsy death was sudden, before venous stasis progressed to that degree.

In my experience young persons who have contracted their aortic insufficiency in consequence of endocarditis rarely suffer from cardiac pain, while, on the other hand, I have observed numerous instances of angina pectoris in individuals whose aortic lesion had resulted from degenerative changes. In 1891 I began to treat a married woman of about thirty who was afflicted with aortic incompetence and attacks of præcordial pain. She was quite stout, and this made examination of the heart difficult. The radial pulse was collapsing, though not as full and quick as in typical cases, and she had an aortic regurgitant murmur. The apex-beat could not be distinctly made out, and the large breast prevented my determining the boundary of deep-seated dulness at the left. The absence of manifest cardiac hypertrophy rather puzzled me, but eventually led me to conclude that the leak was not very free, and consequently that there was not much hypertrophic dilatation of the left ventricle. After she had been under treatment for a time she called attention to a pulsation in the neck. This was found to be just behind and above the right sterno-clavicular articulation in the location of the innominate artery. It was attributed to aneurysm of the arch of the aorta, which thus brought the innominate prominently into view. The patient was taken to several diagnosticians for opinion, and among others to the late Dr. Christian Fenger, by whom my diagnosis was confirmed. This discovery of a probable aneurysm changed my views concerning the etiology and pathology of the case. Whereas the history had led me to regard the aortic regurgitation as of rheumatic origin, I now considered it secondary to aortic aneurysm, a view that seemed to account for the attacks of angina.

The patient then left Chicago, and I did not see her for several years. At length I was one day unexpectedly summoned to visit her at one of the hotels to which she had betaken herself immediately upon her return from Europe the day before. She was in a truly pitiable plight. The attacks of agonizing pain had become so frequent and severe that she literally could not walk across the room without one being evoked, and she was taking large doses of nitroglycerin and whisky, though with but slight effect. The circumference of the neck was greater than normal, although the evident congestion had not produced œdema. The old-time pulsation was still in evidence. It did not appear to have increased in area, and the only alteration I could detect in the heart-findings was greater rapidity and feebleness of action. She was given injections of morphine sufficient to somewhat blunt her sensibility to pain, but aside from this there was nothing that could be done. She dragged out a miserable existence for a few weeks longer, and then in one of her attacks death mercifully ended her sufferings.

At the autopsy, which was performed by Dr. Frank S. Johnson, who had also seen the case, the aortic valves were found very incompetent and sclerotic, but whether the process had originally been of endocarditic origin or not it was difficult to decide. There was a moderate degree of enlargement of the left ventricle, the walls of which were fatty. The two most interesting features, however, were (1) occlusion of the mouths of the coronary arteries by deposits of lime-salts, so that they with difficulty admitted the point of a fine probe, and (2) the size of the aorta. No aneurysm could be detected, but careful measurement showed that the ascending portion of the arch was uniformly increased in diameter by about 1 centimetre, while its walls were possibly a trifle thinner than normal. It seemed probable, therefore, that when distended by the abnormally large blood-wave it became stretched sufficiently to amount to a considerable dilatation, which had caused some pressure on the great veins, hence the congestion of the base of the neck, and the prominence of the innominate artery.

It was now easy to understand the frequency and intensity of her angina. The heart-muscle simply could not be flushed with blood through the extremely narrowed coronary ostia. Whenever physical exertion called for more blood within the coronary arteries it was not forthcoming, and cardiac ischæmia was manifested by a cry of agony.

From the foregoing, it is plain that cases of aortic regurgitation can be divided into two classes. In the one, the lesion is the result of endocarditis, contracted during a period of life when the myocardium and arterial walls are still young and healthy—great compensatory hypertrophy is possible, and the disease may endure for many years without giving rise to symptoms. These appear at length only after the heart-muscle can no longer be sustained by the coronary circulation, or the breakdown occurs as the result of fresh endocarditis ingrafted on the old process in the course of acute articular rheumatism. In my care, five years ago, was a vivacious young lady of eighteen, who presented the typical signs of free aortic regurgitation, a quick, collapsing pulse, a broad, heaving apex-beat, situated far below and to the left of its normal situation, and a loud diastolic murmur. She consulted me because of having noticed that she could no longer run upstairs, dance, ride a wheel, or do other things which before were unattended with consciousness of the heart's action. She did not get out of breath, but was annoyed by forcible pounding of the heart and by the occasional sensation as if it "gave a flop."

She had some flatulent indigestion and was constipated. The pulse was now and then intermittent, and for the purpose of correcting this intermittence she was given small doses of tincture of digitalis, 5 drops 3 times a day. When she next returned, after a few days, she stated that the pounding of the heart was worse instead of better. The digitalis was reduced, but still intensified her symptoms, and was discontinued. Thinking that the intermissions might be due to gastro-intestinal derangement, she was given remedies to correct the constipation and improve digestion. There was some improvement, but still the heart did not become entirely regular. One day she complained of dull frontal headache, some pains and stiffness of the muscles, which seemed to me a muscular rheumatism, possibly of uric-acid origin. Accordingly, she was put upon potash and salicylate of soda, and ordered to drink freely of water. This was a happy hit, for she lost the intermittence of the pulse, and was no longer annoyed by the heart's pounding.

A year later, believing I had discovered evidence of a tendency to growing dilatation of the left ventricle, I gave her a course of Nauheim baths, which agreed with her, and she felt so well that I lost track of her for some months. Indeed, with one exception, after the baths were finished, I never saw her again. But one day, encountering her mother in the cars, I learned that in August, eight months subsequent to her last visit at my office, she developed what appeared to be a mild attack of articular rheumatism. As I was out of the city, a neighbouring physician was given charge of the case, and he very properly confined her to bed. After about a week the rheumatic manifestations had subsided, and she was thought to be getting on finely. One morning she awoke in excellent spirits and seemed nowise in immediate danger. Nevertheless, during the forenoon, when she sat up in bed to drink a glass of water, she suddenly, without warning, fell back upon her pillow and expired. No autopsy was held, and I have no means of knowing the exact condition, but I believe that probably the heart had become weakened by fresh endocarditis attending the mild rheumatic attack, and under such circumstances the exertion of sitting up occasioned sudden diastolic arrest of the left ventricle. It simply illustrates the liability of these patients to sudden, unexpected death.

In the second class belong patients whose valvular defect is the local manifestation of degenerative changes, which, if not due to syphilis, the gouty diathesis, strain, and the like, are associated with advancing age. In such persons compensatory hypertrophy rarely proves so enduring as in the young, and may fail early, because the myocardium is already degenerated, or because the state of the coronary arteries does not permit that degree of nourishment necessary to the maintenance of hypertrophy. In this second class should also be reckoned those cases in which the aortic insufficiency is the result of rupture. In this latter group, pain, præcordial distress, and other symptoms of cardiac incompetence are apt to appear promptly after the injury, and to persist without relief. Naturally, however, the ability of the heart to compensate the defect depends upon the extent of rupture—that is, the degree of regurgitation permitted—and upon the state of the heart-muscle. Dilatation of the left ventricle usually develops rapidly, with little or no hypertrophy, and hence after a few weeks or months the heart succumbs.

In persons suffering from slowly induced degenerative changes symptoms appear slowly, but are never so delayed in coming as in patients whose incompetence originate in endocarditis. In most instances the symptoms that initiate breaking compensation are such as may be referred either to cerebral anæmia—i. e., vertigo and syncopal attacks—or to cardiac fatigue and degeneration—i. e., irregularities of the pulse, palpitation, and angina pectoris.

When, on the other hand, cardiac failure leads to stasis in the lesser circulation, or in the great veins of the general system, the symptoms gradually become those of the terminal stage of mitral disease—i. e., dyspnœa, cough, and frothy, or it may be sanguineous expectoration, disturbed visceral functions in general, œdema, and attacks of threatening asystolism. When at last aortic regurgitation has reached this stage the struggle is less likely to be protracted, and death is usually more sudden than in defects at the left auriculo-ventricular orifice.

Physical Signs.—*Inspection.* — In cases of pronounced aortic regurgitation the disease reveals its presence to the skilled eye by the throbbing of the temporal and carotid arteries. In contrast with the cyanosis of mitral disease the aspect of the patient is apt to present more or less pallor, especially if the disease has developed in early life. Inspection of the chest usually detects strong pulsation of the cardiac area to the left of the sternum, the degree and extent of this pulsation depending upon the thinness and flexibility of the chest-wall, as well as on the hypertrophy of the heart. Occasionally a wave-like impulse is seen to pass from the base downward towards the apex-beat, while in some cases there may be slight systolic retraction of the third and fourth interspaces to the left of the sternum, in consequence of atmospheric pressure, as the hypertrophied heart recedes from the chest-wall.

The apex-beat is displaced outward and downward, in some cases even as far as the seventh or eighth left intercostal space, close to the left anterior axillary line. It is broad and heaving, at once conveying the impression of a large and powerful organ. In the young, with broad intercostal spaces, the dimensions of the left ventricle may be almost as accurately delineated by the visible impulse as by percussion.

In middle-aged individuals, on the contrary, particularly if the chest is capacious, the apex-beat may be scarcely perceptible. In some instances, no doubt, this is owing to the inability of the degenerated heart to establish great compensatory hypertrophy. When very free regurgitation is compensated by great hypertrophy, the eye sometimes discerns visible pulsation in the peripheral arteries, as the radial or the dorsalis pedis. This phenomenon, which is brought out with special distinctness by extension of the hand or foot, is the ocular manifestation of that peculiarity of the pulse about to be described under palpation as the *pulsus altus et celer.*

Quincke has described a visible pulsation of the *retinal artery*, which may be more or less tortuous and elongated with each pulsation. Capillary and venous pulse will be considered later on.

Palpation.—The hand laid upon the præcordium detects powerful cardiac impulse, and over the apex-beat sometimes perceives a short presystolic thrill, or rather receives an impression as if the tip of the heart slid up to its maximum impulse. The impact of the apex resembles the striking of a huge fist against the chest-wall, and if the patient be slight, the whole chest may seem to quiver with the shock. Systolic thrill is sometimes felt in the aortic area. Under some circumstances a diastolic thrill is also manifest.

The most remarkable feature in this part of the examination is presented by the pulse. Its characteristics are so distinctive that a diagnosis is often possible from it alone. First carefully studied and accurately described by Sir Dominick Corrigan, it is often called *Corrigan pulse*, while other terms applied to it are the *collapsing pulse*, the *water-hammer pulse*, the *locomotive pulse*, and the *pulsus altus et celer.* In well-marked cases the finger laid upon the radial artery, or upon any other readily accessible artery for that matter, is suddenly lifted by a large, powerful pulse-wave, which, advancing swiftly along the vessel, strikes the finger like a shot or ball, and then instantly recedes. The vessel, in other words, after being quickly distended as quickly collapses, hence the name collapsing pulse. It is well shown in the accompanying tracing (Fig. 54).

This characteristic of the pulse is intensified by raising the patient's hand to a level higher than that of the heart, and thus allowing the force of gravity to hasten the quick recession of the pulse-wave.

The quickness of the pulse-wave has thus been dwelt upon for the purpose of emphasizing the difference between the speed with which it travels along the artery, and the frequency with which the individual pulse-waves follow each other. Consequently, a frequent pulse is a rapid or accelerated pulse, whereas *a quick pulse is one that strikes the finger suddenly and is not sustained.* A pulse may be both frequent and quick, as in fever, but a quick pulse does not necessarily have to be also a rapid one. In aortic regurgitation, however, the pulse is both sudden and accelerated.

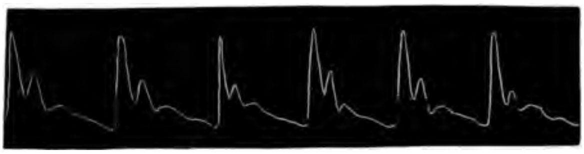


FIG. 54.—SPHYGMOGRAM OF AORTIC REGURGITATION.
Tracing by Dr. Edward F. Wells.

In some cases when the arteries have become more or less sclerotic and tortuous the bounding pulse-wave seems to lift the vessel from its bed, and hence some writers have spoken of it as the *locomotive pulse.* To make the *raison d'être* of this collapsing character understood, it is necessary to describe how the valvular disease under consideration modifies pulse-tension.

Under normal conditions blood-pressure within the arterial system is maintained at a uniform height by the periodic discharge of blood into the aorta and by the elastic recoil of the arterial walls aided by the tightly closed semilunar valves. The blood-stream driven against the valve by the recoiling aortic walls is intercepted and forced onward through the arterial system. If the aortic valves, incompetent by disease, are unable to check the backward flow of the blood a portion of it regurgitates into the left ventricle, and blood-pressure in the arterial system is correspondingly lowered instead of being maintained at a uniform level. Accordingly, the wave of blood constituting the pulse-wave quickly recedes and allows the arterial walls to collapse, as it were. The hypertrophied left ventricle, made more than normally capacious by dilatation, discharges its contents with a degree of energy proportionate to its hypertrophy; and as its contents are augmented over the normal by the amount that has the moment before regurgitated, the aorta becomes powerfully distended by this abnormally large mass of blood. In consequence of the partial emptiness of the arteries caused by the regurgitation the large blood-wave meets with but little resistance, and travelling rapidly towards the periphery, distends the arteries in its course.

Hence the greater the compensatory hypertrophy of the left ventricle, the fuller, stronger, and quicker will be the pulse. The freer the regurgitation the more marked will be the collapse of the vessel-walls. The degree of difference, therefore, between the distention and collapse of the artery is a measure not only of the degree of the regurgitation, but also of the resulting compensatory hypertrophy, for when the left ventricle begins to fail, this peculiar collapsing quality of the pulse grows less pronounced, although the regurgitation is no whit less free.

Very exceptionally the pulse is said to exhibit the character known as bisferiens and represented in Fig. 55. If the finger is pressed lightly on the artery it receives a sensation as if the pulse-wave were divided into two portions, of which the second is the stronger. The former represents the sudden distention of the artery, and the latter is the palpable expression of the prædicrotic or tidal wave. *Pulsus bisferiens* is usually stated to be found in aortic obstruction, but according to Graham Steell, cited by Clifford Allbutt, undoubtedly occurs in some cases of regurgitation associated with little if any stenosis. In one of Steell's instances this peculiarity was not equally constant or pronounced on both sides of the body. Its production is therefore difficult of explanation, as well as inconstant. I have never obtained a tracing showing a bisferiens pulse in aortic insufficiency, but I have certainly felt pulses in some cases which, to my finger, seemed plainly of this character.

FIG. 55.—P. BISFERIENS.
Allbutt's Syst. of Med., vol. v, p. 931.

Not infrequently, pulsation is so pronounced in the arterioles that the fingers of the patient throb appreciably when grasped and the diagnosis of his malady can be made while in the act of shaking his hand.

Two other phenomena, the *capillary pulse* and visible *venous pulse,* should properly have been described under inspection, but have been reserved until now for the reason that they will be better understood after what has just been said concerning the peculiarities of the pulse. In cases in which arterial tension is very low in consequence of free regurgitation, the capillaries are distended by the blood-wave instead of being kept uniformly filled, and hence display what is known as the capillary pulse (Quincke's sign). This may be well seen in the palm and beneath the nails when the hand is warm, or it may be evoked by friction of the skin—e. g., of the forehead—until an area of hyperæmia is produced. If the periphery of such a red zone is closely watched, its edge will be seen to alternately advance with each systole and recede with each diastole of the heart. Capillary pulsation is also sometimes plainly visible on the soft palate.

By *venous pulse* is meant a visible pulsation in the superficial veins. This is sometimes well marked in the subcutaneous veins of the back of the hand and the forearm when the extremity is allowed to hang down until the vessels become turgid. This venous pulse is a slow undulatory wave which, as Broadbent suggests, may be best noticed by laying a filament of sealing wax across the surface of the vein. Venous pulsation is specially pronounced when arterial tension has been still further reduced by fever. Neither of these last two phenomena is peculiar to aortic regurgitation, for they may be observed in severe anæmia which has sufficiently lowered pulse-tension. They are, however, most distinct and typical in aortic incompetence.

Finally, when regurgitation is very free, a distinct thrill may be felt in the cervical arteries and even in the brachials. This was well felt in a man of about thirty-five, who died suddenly a few weeks subsequently. In this case the thrill was palpable when the finger was laid ever so lightly on the vessel, and seemed to be but the palpable expression of vibrations imparted to the arterial coats by the suddenness and violence of the impact of the blood-stream.

Percussion.—As in other cases of valvular disease, percussion affords our best means of noting to what extent and in what direction the heart has suffered enlargement. It is particularly valuable in cases in which the size of the chest or the feebleness of cardiac impulse prevents us from judging of the size of the heart by inspection and palpation. In compensated cases cardiac dulness is increased only to the left and downward, and the outline of the left ventricle is rather pointed (Fig. 56). As dilatation comes on, the left cardiac border becomes more rounded and the apex is blunt and broad, so that one should always strive to percuss out the shape of the left ventricle as well as its distance from the median line (Fig. 57). Increased dulness to the right is present only secondarily, and is a measure of back pressure important to determine.

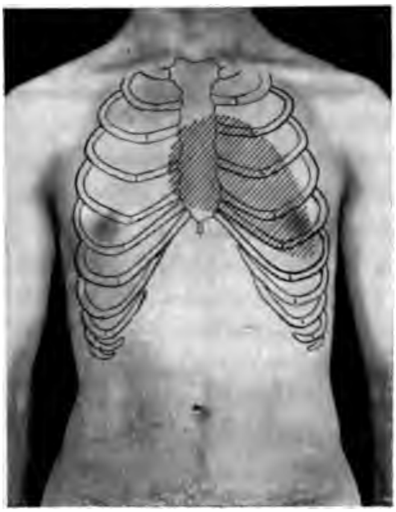


FIG. 56.—TYPE OF RELATIVE DULNESS IN WELL-COMPENSATED AORTIC REGURGITATION.

Auscultation.—Regurgitation through the aortic valves declares itself by a murmur synchronous with the second heart sound and therefore diastolic in time, which is heard with greatest intensity over the base of the heart anywhere between the second right costo-sternal articulation and the junction of the fifth left costal cartilage with the breastbone (Figs. 58 and 59). Its most usual seat of maximum loudness is on the body of the sternum at the level of the third costal cartilage, and yet in some instances it may be heard most plainly or heard

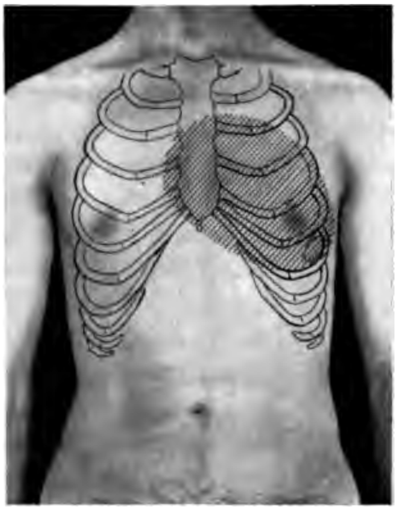


FIG. 57.—TYPE OF RELATIVE DULNESS IN POORLY COMPENSATED AORTIC REGURGITATION.

only in the fourth left interspace, close to the breastbone. It is generally most distinct in the erect position or when the heart's action is excited. Nevertheless I have certainly observed cases in which the murmur became more distinct and easily recognised when the patient was recumbent. This murmur is transmitted downward towards the ensiform appendix, and in some instances also towards the left, even as far as the apex. When audible, with more than usual intensity at the apex, the murmur is thought by some to indicate incompetence of the left posterior flap.

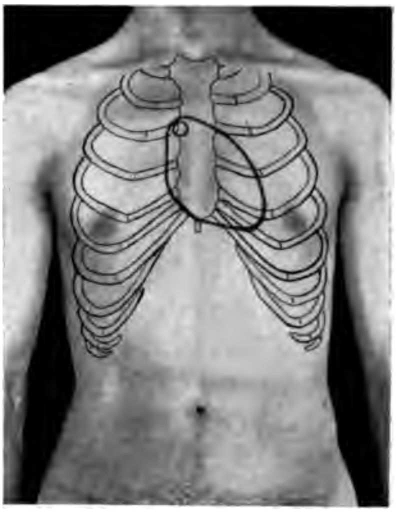


Fig. 58.—Spot of Maximum Intensity (small circle) and Area of Transmission of Typical Aortic Regurgitant Murmur.

As previously remarked with reference to the mitral regurgitant murmur, the intensity and the extent of conduction of this aortic diastolic murmur furnish no criterion of the gravity of the lesion. Indeed, numerous instances have been recorded in which no bruit at all was audible for a variable time immediately

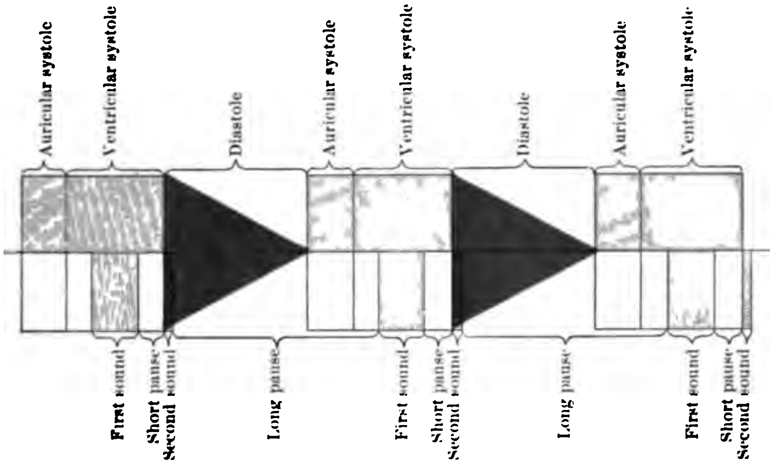


Fig. 59.—Rhythm of Aortic Regurgitant Murmur.

prior to death. This is probably owing to a want of sufficient force and rapidity in the regurgitant stream to generate soniferous vibrations. The duration of this murmur is usually short, and its quality is soft rather than harsh, and is unlike that of any other murmur excepting the bruit of pulmonary regurgitation.

In exceptional cases the diastolic aortic murmur may have a true musical tone. I well recall the case of a coloured man, in whom the intra-vitam diagnosis of aortic regurgitation was substantiated post mortem, and who presented this musical quality in a most marked degree, but not with every ventricular diastole. At irregular intervals the soft diastolic bruit was associated with, or replaced by a musical murmur so intense that it was heard by the patient, and imparted a distinct thrill to the hand laid upon the heart to the left of the sternum. A few days before death this musical murmur entirely subsided, and at the post-mortem examination no condition that could explain its production could be discovered, although diligently sought for. The valves presented the appearance ordinarily found in cases of endocarditic insufficiency.

The heart-sounds usually present more or less modification. The first sound at the apex is apt to be muffled or toneless, while the second sound is enfeebled. In the aortic area the second sound may be entirely wanting, being replaced by the diastolic murmur, or there may be a faint rudimentary second sound. The aortic first sound may be audible or replaced by a rough, more or less intense, systolic murmur. This bruit is usually interpreted as signifying an associated stenosis. This conclusion, however, is not always justifiable, since such a systolic murmur may be due either to roughness without narrowing of the orifice, or to sclerosis of the aortic intima. When both a systolic and diastolic murmur are heard, they are often spoken of as a " to and fro " murmur, and such a combination is very frequent.

There are also certain auscultatory phenomena connected with the peripheral arteries which furnish valuable secondary signs of this valvular lesion. Over the carotid and subclavian arteries a faint systolic murmur is often found to replace the first sound, while if the regurgitation is free the normal second tone is absent. When one auscultates the femorals, he hears a sharp snap, which is synchronous with ventricular systole and is the audible expression of the sudden tension into which the arterial coats are thrown as they are distended by the large sudden pulse-wave. If rather more pressure is exerted upon the vessel by means of the stethoscope, this snapping tone disappears and becomes replaced by a distinct murmur, the murmur of constriction, which can be elicited over any artery of sufficient size when no valvular disease exists. When regurgitation is free, it is usually possible by trying different degrees of pressure to at length bring out more or less clearly not only this systolic murmur, but also a diastolic one, so that one becomes conscious of a double murmur, of which, in my experience, the systolic is usually the louder. This double femoral bruit was first described by Duroziez, and hence is often spoken of as *Duroziez's sign.* It is considered pathognomonic of aortic regurgitation, since in no other disease are the conditions presented for its production. The explanation of this phenomenon is as follows:

Constriction of the artery throws the blood-stream into audible vibrations as it passes the point of pressure, and, normally, this is all; but in aortic insufficiency the blood-wave recedes during diastole and passes again this point of constriction, with the result that it is a second time thrown into vibrations, and a diastolic murmur is generated. In most cases these acoustic phenomena are elicited only over arteries of large calibre, but when the lesion is very pronounced and the left ventricle is powerful, both the systolic snap and the double bruit may be heard in small vessels, as the radial and even the dorsalis pedis.

Diagnosis.—Ordinarily the recognition of aortic insufficiency is not difficult. In some instances it may be detected at a glance, but when the individual is past middle age, with sclerotic arteries and a voluminous thorax, the collapsing character of the pulse and a powerful cardiac impulse may not be pronounced, and care is requisite to determine that the condition is not an aortic aneurysm that has led to regurgitation. In all doubtful or indistinct cases particular study should be given to the vascular signs, since they are conclusive, and a diastolic bruit is not. Indeed it is to be remembered that when in the last stages the heart has become very weak, the murmur previously present may entirely disappear. Moreover, the diagnosis of this lesion may be rendered not easy by the association of relative mitral incompetence, or of other organic defects. In all such cases one must minutely investigate the vascular system and rely on its disclosures rather than on cardiac findings, although even here valuable information may be obtained if attention is paid to the secondary changes instead of the auscultatory findings.

Before leaving the subject of the diagnosis of this disease, I desire to dwell for a few moments on a subject which has given rise to much controversy. Many years ago the late Austin Flint, one of the most careful clinical observers this country has produced, directed attention to the presence of a presystolic apex-murmur in some cases of aortic regurgitation, and declared it was an accidental murmur which did not necessarily denote the coexistence of mitral stenosis. He was vehemently attacked by Balfour, who declared a functional presystolic murmur an impossibility. But corroboration of Flint's observation has come from so many sources that there can no longer be any doubt of the correctness of his statements.

His explanation of the mode of its production is, however, probably not correct in the light of more recent physiological knowledge concerning the closure of the mitral valves. The murmur is now thought due to vibrations of the mitral curtains as they are caught between the regurgitant stream on the one hand, and that pouring out of the left auricle on the other. It is quite possible for mitral constriction and aortic regurgitation to coexist, and in any instance of this latter disease in which a mitral presystolic murmur is recognised its correct interpretation is made possible by giving due consideration to the presence or absence of secondary changes in the right ventricle, and the smallness yet collapsing character of the radial pulse.

Prognosis.—A proper estimation of the prognosis of aortic regurgitation requires a sharp distinction between the forms due to endocarditis and those of degenerative origin. Furthermore, in each group and in each individual instance, the prognosis is in direct relation to the degree of compensatory hypertrophy, the same as in any other valvular defect. If, in the first class—that is, of endocarditic origin—compensation becomes once well established, it is possible for the disease to be borne for many years. Mr. W., the description of whose sudden death will be narrated, was known to have aortic regurgitation of severe type for at least twenty-eight years, and perhaps longer. When, however, compensation begins to fail, the prognosis is very grave, for it cannot be so readily restored as in mitral disease. Indeed, some authors are of the opinion that compensation can never be reinstated; at the most there being hope only of retarding the downward progress.

When in the second category of cases, those of degenerative nature, compensation becomes established, it is at the best only for a comparatively limited period, owing to the probable presence of chronic myocarditis, and when this compensation once breaks, it is irretrievably gone. Henceforth the progress of the malady is for the most part steadily downward. In all cases the prospect of even partial recovery is slight, and of restoration to a life of activity and freedom from symptoms is nil.

It is stated that very rarely a regurgitation may be converted into a predominating stenosis by the growth on the valves and ring of vegetations, in consequence of fresh endocarditis; and whenever this occurs the prognosis becomes more favourable, provided, of course, there be no myocarditis or other complications. This possibility is too remote to be ordinarily taken into consideration.

Mode and Causes of Death.—No other valvular disease so often terminates abruptly. The suddenness of the death is due to paralysis of the left ventricle in diastole. In most cases, no doubt, warning has been given of the pending catastrophe by irregularities of the pulse, vertigo, or other symptoms which failed at the time to attract the patient's attention, or were too insignificant to impress him with their gravity. Death follows some sudden effort, as assuming the erect from the recumbent position, springing out of a chair to leave the room, jumping on to a moving street-car, and the like. The muscular effort incident to such sudden movements abruptly raises blood-pressure within the vessels supplying the groups of contracted muscles, and drives the blood into the left ventricle during its period of relaxation with a degree of force which the ventricle is unable to resist. It fails to respond by a subsequent systole, and the patient falls to the ground in an attack of fatal syncope.

Fortunately for the patient, as well as for the peace of mind of his friends, assurance can be given that sudden death in the way just described is not usual. Indeed, it occurs in the minority of cases of aortic regurgitation. According to Broadbent, it occurred in 10 out of 38 cases taken from the records of St. Mary's Hospital. Sudden death occurs by far the most frequently as result of fibroid and fatty degeneration of the myocardium, and, therefore, we should look for it in individuals whose aortic valves are incompetent in consequence of degenerative changes rather than in the young, whose valvular lesion is of endocarditic origin. This does not apply, however, when the heart is freshly attacked by an acute endocarditis. The case of the young lady of eighteen illustrates that under such circumstances the end may come unexpectedly and without warning.

We have seen in most cases of aortic regurgitation that the symptoms showing complete loss of compensation become those of pronounced venous stasis, the same as in the last stage of mitral disease. It is to be expected, therefore, that exitus lethalis should take place in the same manner, and in fact such is the case—i. e., from gradual cardiac asthenia or acute pulmonary œdema.

Strümpell directs attention to the not infrequent occurrence of pericarditis in aortic insufficiency, particularly when the valves have been attacked by fresh inflammation, and in such the pericarditis is very apt to lead to the death of the patient.

Of 24 cases analyzed by Hustedt, the causes of death were as follows: Heart-weakness, 8; pulmonary infarct, 9; pneumonia, 2; œdema of the lungs, 3; apoplexy, 1; and pleuritis, 1.

The suddenness of death is illustrated by the case of Mr. W., aged forty-five, who had had aortic insufficiency dating from chorea and rheumatism in boyhood, and had shown symptoms of failing compensation for at least two years. These consisted in feebleness and irregularity of the pulse, and attacks of weakness and faintness of such severity that they compelled him to seek aid of the nearest physician. Notwithstanding the extreme degree of fatigue and exhaustion occasioned by his duties, he persisted in the daily attendance at his office. The day of his death he left home as usual and proceeded to his place of business. While in the act of stooping over a table, he sank to the floor, and exclaimed, " I am dying! " His clerk, who was standing near, lifted him into a chair, and asked if he should run for a doctor. The sufferer looked up, smiled, and shook his head, as much as to say, "No, it is of no use," and a few moments thereafter he quietly expired. That this patient was fully aware of his being in daily—yes, hourly—danger of death was shown by the fact that he had given explicit directions to his clerk what to do in the event of such a fatality. Reflecting upon this case, one cannot help wondering by what knowledge this patient recognised the significance of his final attack, and refused to have a medical man summoned, when in previous attacks he had always sought the services of the most accessible physician.

The following case is appended because it emphasizes in an impressive way several points with respect to aortic regurgitation. In the first place it illustrates the important part played by heart-strain; in the second, how hopeless is the prognosis in these cases; thirdly, the futility of treatment; and, lastly, the manner of death in a considerable proportion of them.

I recently witnessed the death of a gentleman of forty-two who had sought medical advice three months before on the supposition that his distress was due to some form of stomach trouble. Excepting scarlatina at the age of six, he had never known a day's illness, and his habits had been exemplary with the one exception that he had been accustomed to take about two drinks a day of whisky before meals. He had always been devoted to hunting and fishing, and had spent much time each year in the woods, at which times he had always shouldered his pack and tramped along with his guides, having many a time, as he said, "Done them up and come in at night fresh as a daisy, while they were beat out." He had thought nothing of carrying 60 pounds on his back all day through the woods, and had paddled and portaged with the best of them. When not out hunting or fishing he had been untiring in his devotion to business, and for the previous seven years had worked with colossal energy in building up vast interests in the North. In addition to tireless work with his brain in his office, he had endured and indeed revelled in efforts connected with his business schemes, requiring and displaying extraordinary physical endurance, so that he was the marvel of his friends. On one occasion, in December, 1900, this robust man of medium stature and weight, without an ounce of superfluous fat, all muscle and sinew, started with a crew of men up his railroad to inspect some work, and, as the engine was forced to "buck snowdrifts" and make a way for itself, the engineer suddenly discovered that, having neglected to fill his tank afresh, it was out of water. The weather was intensely cold, and this meant that the fire would have to be dumped and the locomotive be allowed to freeze up, or that in some way a supply of water must be obtained. They were near a river, and this indomitable man of whom I write started on a dead run through the deep snow for a camp of his men a mile and a half distant, where he knew he could obtain some pails. He went himself because he knew he could get there in quicker time than any of his men, and, too, would have the authority to take the buckets. Arriving there, he shouldered a package of half a dozen iron buckets and started back, running all the way through the snow with his load of 75 pounds. He arrived in time to save the engine, but completely exhausted. Nevertheless he recovered in the course of the day, and thought nothing more of it, going about his herculean daily work in and out of the office as before. But outraged Nature was to have her revenge yet. In April following his almost superhuman effort, this man of affairs took a hard, fast horseback ride of ten miles over a very rough road, and before he reached his destination he became seized with a severe pain in the epigastrium, which, however, ceased to trouble him greatly after he had dismounted. From that time on until I saw him the next October, he had grown steadily less and less able to endure exertion without this epigastric distress, to which dyspnœa finally became added. In June following his ride, he had climbed down and up a ladder into and out of a mine several hundred feet in depth, and on reaching the surface again had noticed that he was very much winded, and from this time forward he was obliged to walk slowly if he did not wish to suffer from his pain and shortness of breath. The night previous to his arrival in Chicago, and on several other occasions, he had been awakened in the small hours by a feeling of oppression which compelled him to sit up on the edge of his bed and breathe hard. On this particular occasion he had attributed his attack to the closeness of the sleeping-car, had taken a drink of whisky, experienced speedy relief, and, lying down, had gone to sleep. Such in brief was his history, as full of interest as a romance.

From his recital I expected to find a case of simple cardiac dilatation from overstrain, such a case as I had shortly before finished treating. Imagine my surprise, therefore, when I discovered a collapsing but not large pulse of about 110, a diffused, rather indefinite apex-beat way below and outside the left nipple, percussion evidence of a greatly hypertrophied and dilated left ventricle (Fig. 60), feeble heart-sounds, and everywhere a loud double murmur plainly aortic in origin. Duroziez's sign and capillary pulse were present, and the liver was palpable and tender a couple of inches below the inferior costal margin. The left lobe was specially swollen and sensitive to pressure. The urine was and always remained negative.

Here, then, was an aortic regurgitation, but what was its etiology? Was it possible that there had been a valvulitis years before and that the compensation had been broken down by his prodigious exertions, or had the heart-muscle been not quite healthy and had that run started a stretching of the aortic ring which had been increased by succeeding efforts, or had the strain led to an aortitis or aneurysm, and this to insufficiency of the valves? Rupture of a cusp was out of the question, because of the absence of serious symptoms in the weeks immediately succeeding his run. A previous valvulitis was not impossible, for it is well known that the enormous secondary hypertrophy of the left ventricle sometimes developed in cases of rheumatic incompetence of the aortic valves, is capable of enduring an extraordinary degree of strain for years, as witness some of the cases treated by the great Stokes. In this instance there was no history of anything to lead to endocarditis except the scarlatina, and that occurred thirty-six years before, and if that had led to valvular insufficiency its presence had never been suspected or betrayed by a symptom. Moreover, thirty-six years is a very long time for an aortic regur-

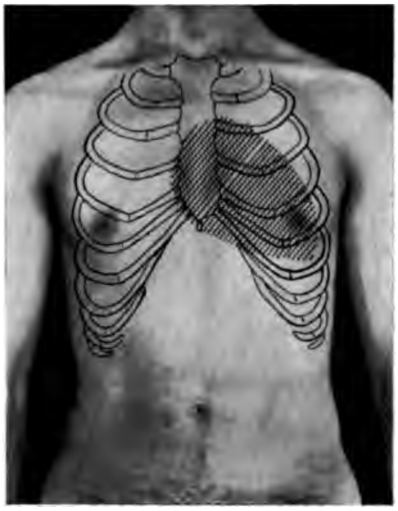


Fig. 60.—Relative Dulness in Case of Aortic Regurgitation (p. 309).
First examination.

gitation to exist without discovery. I therefore considered this explanation as less likely than one of the others. Regarding aneurysm as cause of aortic incompetence, I had already observed a case in which such was the condition, not a very unusual one, but for the greater part of a year there had been no symptom to point to aneurysm, and it was not discovered until three months prior to death from pressure on the left lung. Consequently I now had a Roentgen-ray picture taken for the detection of aneurysm if such existed. It is shown above (Fig. 61), and shows great breadth

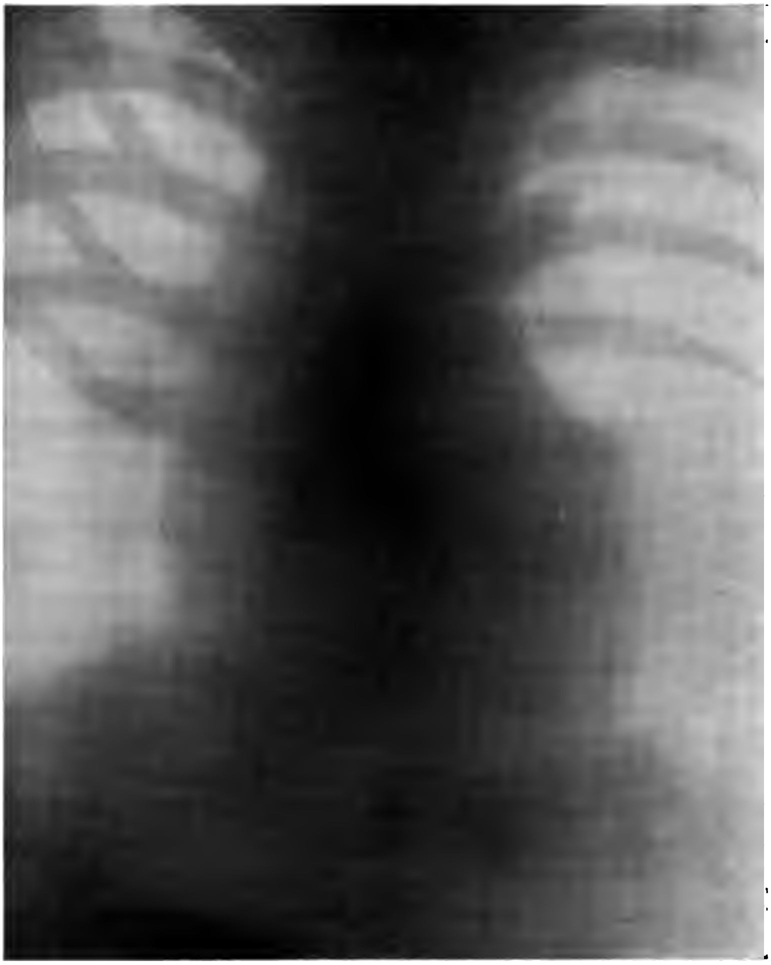


FIG. 61.—SKIAGRAM OF CHEST IN CASE OF AORTIC REGURGITATION.

of shadow at the base over the position of the large vessels, but nothing that can be interpreted to indicate aneurysm. It also shows a very large heart, a veritable *cor bovinum.* The only remaining hypothesis was that of stretching of the aortic ring and base of the aorta. The vessel could be felt pulsating at the level of the upper edge of the sternum, and in the aortic area could be heard a faint distinct click, evidently a feeble aortic second tone.

Another case that has been already narrated (see page 158) had taught me how guarded one should be in attributing to endocarditis what might turn out to be attributable to myocarditis and stretching of the orifice, especially in the absence of a definite history of rheumatism or other sufficient etiological factor. In the present instance this point was of great importance in its bearing on prognosis. I believe the subsequent lack of resistance on the part of the left ventricle bore out strongly my original view of the origin of the valvular incompetence. The leak was started by the awful strain of that insane run, and was augmented and rendered hopeless of repair by his succeeding exertions.

The man was told how serious his condition was, and that his only hope of reinstating his heart-power lay in at once giving himself up to entire and prolonged rest in bed. Very reluctantly he yielded to the inevitable, and took to his bed. He was of a somewhat peculiar nervous make-up and could not be induced to remain as inactive as I thought was necessary. He would, for instance, get up and go to the toilet in the adjoining bath-room instead of using a bed-pan. He would get up and shave himself, and he would sit up in bed to eat, and on one or two occasions arose to receive a visitor. I now blame myself for having permitted even so much latitude, yet his annoying symptoms disappeared so promptly on being put to rest and his left ventricle came down so appreciably in size and the apex-beat returned in such strength, that I hoped, against my first judgment, that the heart was going to recover its hypertrophy better than was at first feared.

At the end of a month of this enforced inaction the patient became so restless that permission was given him to leave his bed and, by degrees, begin to move about, under the condition that he was to lie down much of the time. He was allowed also to take a daily drive. A course of Nauheim baths was also begun in the hope of re-enforcing the effect of the digitalis, nitroglycerin, and strychnine he was taking. For a week he did not seem to suffer any ill consequences, although he ignorantly overdid in various, to him, seemingly trivial ways. He would put on his heavy fur-lined overcoat unassisted, and go into a store to make purchases, and be on his feet for an hour at a time, not realizing that he was still far from well. Then the saline baths did not slow his pulse and improve its quality as they should have done, and after a bath he did not react to my satisfaction. So after two weeks, in which it was clear that he was losing ground, I again ordered him to bed, this time insisting on his having a trained nurse who was to lift him, and in many ways save him from efforts he had made during the earlier weeks. He now remained fairly quiet, though it was very difficult for him to learn to keep still and not to assist his nurse whenever she lifted him, turned him, etc. He simply would not use a bed-pan, and therefore was drawn in a chair to the bath-room, and later on was lifted on to a night-stool alongside his bed. His treatment consisted of digitalis and other tonics, cathartics, and resistance exercises given by a competent attendant. Although it was realized that these last were in violation of the principle of absolute physical repose, still they were decided on because they slowed the pulse somewhat, improved its quality, and so quieted him that he fell asleep after the séance was over. No more Nauheim baths were given.

As days ran into weeks, however, it became plain to me that treatment was not going to restore what he had lost during the two weeks he had been up. Indeed, he slowly but perceptibly lost ground. His liver swelled again somewhat and gave a very uncomfortable feeling of fulness below the ribs, which he attributed to his stomach and to indigestion. The outline of the left ventricle very gradually became more rounded, the apex less pointed, and its impulse less vigorous. It was at length clear that if the mitral valve was not already relatively incompetent, a matter for certain reasons difficult of positive determination, it would soon become so.

January 1st he was moved from the hotel into a rented house, and the removal was effected as easily and with as little disturbance to him as possible. Nevertheless when I visited him a few hours later I saw at once that the transfer had hurt him. His pulse was less strong, the heart-dulness a little larger, and his breathing a little less easy.

The feature that especially increased my anxiety, however, was the peculiar irregularity of the pulse. At varying intervals, from 5 to 20 beats, there would come a sudden quick wave closely following the one before, as if the heart were trying to catch up in its work by giving an extra contraction (*pulsus intercurrens*). The patient, moreover, appeared totally unconscious of this peculiar action, which did not disappear during the remaining two weeks of life until the dilatation of the left ventricle had grown so extreme as to lead to relative incompetence of all the other valves. One night, apparently in consequence of flatulent distention of the bowels, he became extremely nervous and alarmed over a fluttering of the heart, which persisted for several hours, and until after a dose of whisky administered by the nurse he fell asleep.

To add to the damaging effect of all this strain, the patient became nervous and despondent, and so desirous of getting out of doors that I agreed to his going out in his wheel-chair provided he was carried downstairs on a stretcher, then placed in his chair, and on his return brought up again in the same manner. This was attempted but was bungled in some way so that he was rendered extremely nervous, and, after all, was borne down and up in his attendant's arms. This in reality ought not to have injured him had his heart-muscle been less seriously damaged. As it was I recognised, so soon as I examined him a short time afterward, that the walls of the cardiac cavities were still more stretched and the liver still more engorged.

He now lost his appetite entirely, passed rather poor nights, and showed a slight puffiness of the insteps. One week later he began to have very slight nausea, and on Sunday forenoon was seized with a sudden attack of vomiting. He rose up in bed and strained violently in the act in a way to make me most uneasy. I happened to be sitting by his bedside at the time and took occasion to observe the pulse. This did not become specially accelerated but rather thready, and its irregularity somewhat more pronounced. Examination of the heart did not, however, reveal any marked ill effect. He was now put on kumyss, and all internal medication was stopped lest the stomach might be again disturbed. He passed a poor night and the next day complained of rather more fulness in the epigastrium. About noon this feeling of distress increased, a " hard lump " appeared above the umbilicus, and about 4 P. M. he had a prolonged nervous chill. They succeeded in reaching me by telephone at this time, and I ordered an injection of morphine $\frac{1}{8}$ with $\frac{1}{150}$ of atropine, thinking, as considered by the nurse, that the pain and " knotting " might be an accumulation of flatus in the colon. Upon arriving an hour later and examining the abdomen, I at once discovered that the left lobe of the liver was greatly swollen, tender to pressure, and was throbbing from the propagated pulsation of the aorta beneath. It required only a brief examination of the heart to perceive that the strain of vomiting the day before had done its evil work by setting up a marked increase of back-pressure (Fig. 62). The external jugulars were swollen, the right heart more dilated, and the pulmonic second tone very muffled. A cathartic was ordered and he was at once put on a hypodermic of nitroglycerin $\frac{1}{100}$ with $\frac{1}{60}$ of strychnine sulphate every four hours. Half a dozen watery stools the next morning made him feel comfortable, but it was clear that back-pressure was on the increase. By noon he was quite cyanosed, and the pulse was small and weak. He was then put on 15 drops of fat-free tincture of digitalis every two hours, the glonoin, strychnine, and cathartics being continued.

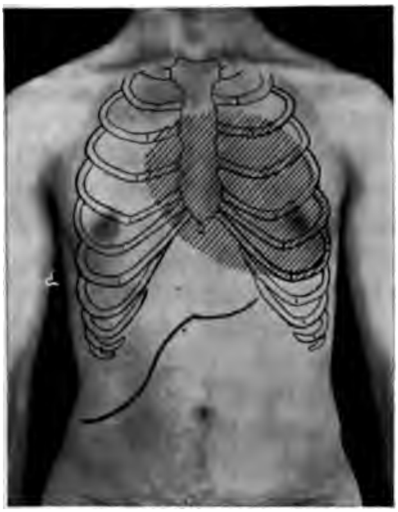


FIG. 62.—RELATIVE DULNESS AND LOWER BORDER OF LIVER SHORTLY BEFORE DEATH. Same case as Figs. 60 and 61.

Wednesday morning, in spite of free hydragogue catharsis, his condition was still worse. The pulmonic second tone was replaced by a soft diastolic murmur, and he was cyanosed. Even turning him in bed produced profound cyanosis, feebleness of the pulse, and difficulty of breathing. The morphine was continued hypodermically in doses of $\frac{1}{8}$ once or twice in twenty-four hours to prevent restlessness and unnecessary suffering, while the interval between the injections of nitroglycerin and strychnine was shortened to two instead of four hours. He then rallied for a few hours and the cyanosis almost disappeared, but the heart-findings remained about as before. Morphine gave him a fairly comfortable night and Thursday came. The pulmonic second sound was now audible again, but the pulmonic diastolic murmur persisted and the external jugulars and liver pulsated unmistakably, while a soft systolic bruit, evidently tricuspid, could be heard at right of the sternum near its extremity.

The pulse now began to slow down, from 96 to 90, then to 88, and by noon to 80, yet did not grow stronger. On the contrary, it seemed to grow smaller and weaker, while the jugular pulsation increased, and distention of the right auricle caused absolute dulness to reach across the sternum and beyond. He had now received 24 doses of digitalis, and believing that it would only do still greater harm, I ordered it stopped and provided elixir of valerianate of ammonia and a 10-per-cent solution of camphor in sterilized olive oil against possible further sinking of the pulse. The oil was to be injected under the skin in case of need.

The feet and ankles were now quite œdematous, urine was very scant, the patient perspired profusely, slept much of the time, and was profoundly cyanosed with marked puffiness of the neck and lower part of the face. More bowel movements of a watery character were secured without any impression on the stasis. The pulse stayed at 80, very weak, and whenever to rest him he was turned on to his back or left side, became distinctly worse. His greatest comfort was when he lay in the right lateral decubitus. His respirations were 28, and his breathing was laboured at times. Râles of hypostatic congestion were audible at the right base behind.

In this condition things remained until 10.30 P. M., when suddenly he complained of inability to breathe, turned purple in the face, grew rigid and pulseless. The nurse hastily injected the camphorated oil as I entered the room and hurried to the bedside. I listened for the heart-sounds, but all was still; life was extinct, his muscles relaxed and his sufferings were at an end.

This case has been detailed at this length in the belief that it might prove highly instructive on many of the points that have been already dwelt upon in regard to aortic regurgitation, and will be dwelt on in considering the prognosis and treatment of valvular disease in general.

It brings out only too clearly the utter hopelessness of the prognosis in aortic insufficiency in men of middle age when compensation once gives way, no matter what the cause of the valvular defect. The heart-walls are too degenerated to retain any temporary improvement that may follow appropriate treatment.

In the matter of management nothing is so important as rest, and this should be as absolute as possible. One cannot refrain from looking over his management of a case and being inclined sometimes to upbraid himself for not having done this or that. In this case I now think I should perhaps have been rather more energetic with digitalis from the very start than I was, and yet at the time I feared its effects on the vascular system might offset that on the heart. In another case I believe I will try pushing the drug to the limit of its usefulness. Then as to the degree of rest which was secured. I might have been less lenient, and yet this gentleman had been so active a man that rest in bed chafed him. It was a nice question to decide whether, as a matter of fact, strictness in regard to complete physical inaction would not have made him so impatient and restless in spirit as to have entirely counteracted the benefit to be had from rest of body, or as to have done him greater harm than did the little exercise he took during the first month of treatment. At all events this was how I looked at it then, and I am not sure but I was right. As a matter of fact the result was inevitable, and no treatment could do more than retard the fatal issue.

CHAPTER IX

AORTIC STENOSIS

This is a comparatively rare affection when existing alone, and, as its name signifies, consists of a narrowing of the aortic ostium. It is aways a structural defect and owes its origin chiefly to inflammatory changes.

Morbid Anatomy.—There are two types of structural change that may lead to narrowing of the aortic orifice: (1) The cusps of the valve may become adherent and stiffened; (2) growth and organization or calcification of vegetations may take place in such a way as to interfere with the passage of the blood-stream. The former defect may be due to a developmental error, and occurs in congenital narrowing. It may also, however, follow acute endocarditis. The cusps may become so completely adherent that only a small opening, scarcely large enough to admit the point of a lead-pencil, is left. Fig. 63 shows such a heart, and also illustrates the proneness of acute endocarditis to attack a valve already the subject of chronic disease. Here a tiny row of vegetations is seen along the line of maximum contact during the closure of the valves. In those cases of stenosis of sclerotic origin in which the cusps are not adherent but interfere with the blood-flow on account of stiffness which prevents their swinging back in the normal way, regurgitation is usually so freely permitted that the case is classed as one of insufficiency.

In the second class, vegetations on the aortic valve may assume such proportions as to induce narrowing of the orifice. These occur most usually on the ventricular surface of the valve segments, and in that situation of course interfere also with the closure of the valve, producing leakage. The narrowing is usually the predominant effect, however, of a large vegetation in this situation. An interesting type of stenosis, shown well in Fig. 64, is that in which vegetations develop in one or more of the sinuses of Valsalva. Calcified thrombi in this location almost completely prevent the opening of the valve, and may produce an extreme grade of stenosis.

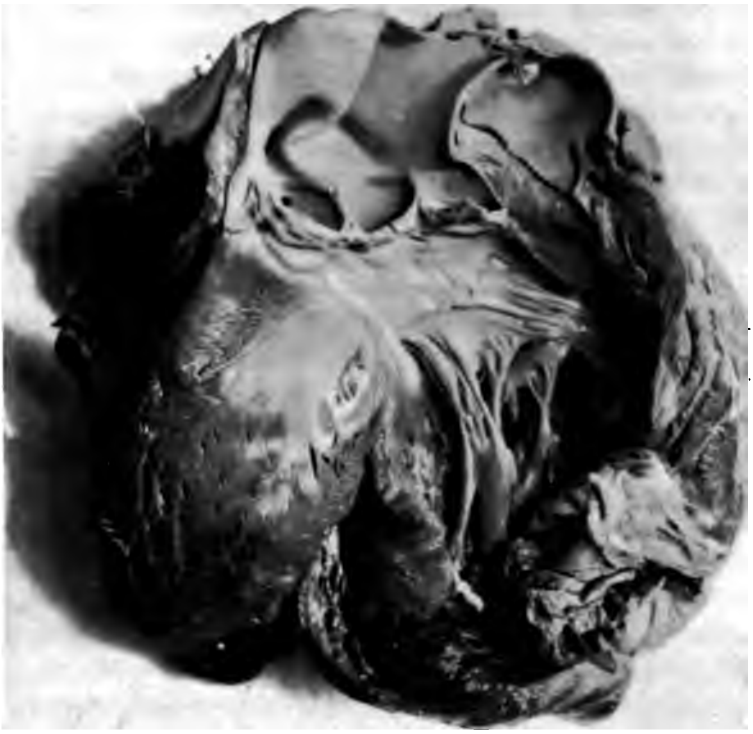


FIG. 63.—HEART OF AORTIC STENOSIS, WITH ADHERENT AORTIC CUSPS, AND ALSO ACUTE ENDOCARDITIS.

Aortic stenosis is not always located in the valve, however, for the aortic ring, and sometimes the whole trunk of the vessel, may be narrowed. This is probably most often a congenital defect. Stenosis of the heart, or more properly of the conus arteriosus of the left ventricle, may also produce the secondary effects of stenosis of the orifice.

It goes without saying that aortic stenosis, according to its degree, presents more or less resistance to the outflow of blood from the ventricle. In order, therefore, to discharge a normal volume of blood through the diminished opening, the ventricle is obliged to contract more powerfully and more slowly. This increased work results in the development of hypertrophy.

Fraentzel is of the opinion that dilatation of the ventricular cavity precedes the hypertrophy, because the wall cannot accommodate itself to its increased task. This would be the case, doubtless, were the stenosis suddenly developed, but inasmuch as the changes in the valves leading to stenosis are brought about slowly, the wall of the left ventricle is able, *pari passu*, to meet the growing resistance. It seems clear, therefore, that hypertrophy of the left ventricle is the first result; and that when dilatation of its

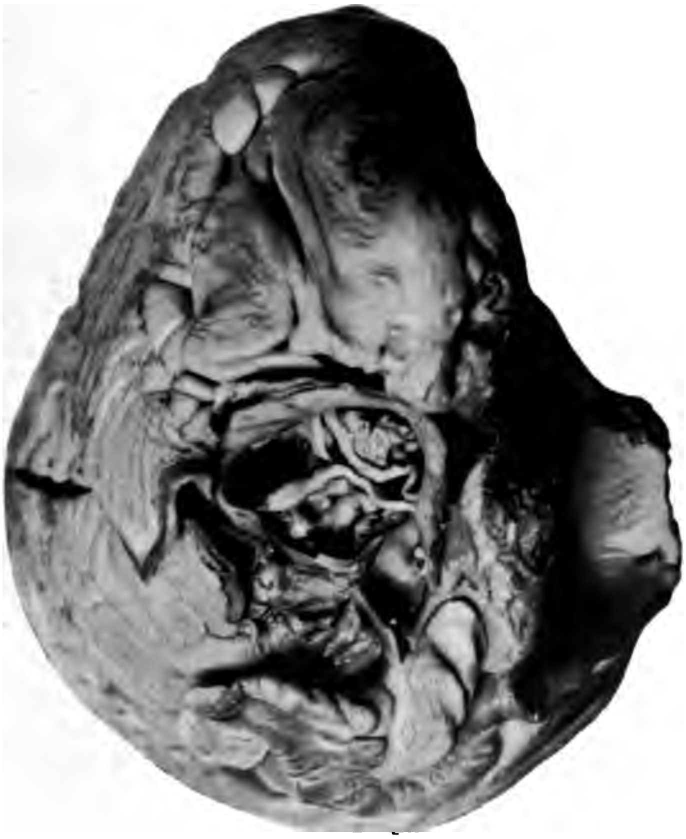


FIG. 64.—HEART OF AORTIC STENOSIS, SHOWING CALCIFIED VEGETATIONS IN SINUSES OF VALSALVA.

cavity is also present, it is either the effect of associated regurgitation or comes on gradually with failing compensation.

Until compensation does fail the secondary effects are limited to the left ventricle. When, however, the ventricle becomes unable to fully empty itself at each systole, the residual blood forms an obstruction to the complete emptying of the auricle. The stasis thus produced creeps back in the manner already described in preceding chapters, with the result of general cardiac enlargement, and the manifestations of passive congestion in the various organs of the body.

In extreme grades of stenosis the supply of blood to the coronary arteries may be so reduced as to cause degeneration of the myocardium. In this manner the constriction tends ultimately to the destruction of that compensatory hypertrophy by which alone the effects of the stenosis can be offset. It will be readily seen that in such a heart as that of Fig. 64, this factor would be of great importance.

Etiology.—Stenosis of the aortic orifice is in nearly all instances acquired after birth and is then due either to endocarditis or sclerosis. The disease occurs more often in the male than the female sex, the same as aortic regurgitation, and, in my experience, more frequently in the young. It is, however, so rarely observed independently of incompetence that of several hundred cases of valvular disease of which I have records, there are only half a dozen instances of pure and uncomplicated stenosis of the kind under consideration. This is readily understood when one reflects for a moment upon the conditions which are responsible for the stenosis.

In those cases in which the valve-segments are agglutinated and rigid, projecting like a cone into the lumen of the artery, there is left a small opening at the extremity of the cone through which a certain amount of reflux is possible. In other cases in which vegetations cause obstruction there is usually such a condition of the thrombi or of the valve-flaps as prevents their perfect coaptation, and hence regurgitation takes place.

When in any given case regurgitation is not also recognised clinically, the conclusion is reasonable that either the valve is not too rigid to close the ostium, or that the seat of obstruction is in the ring or conus.

The etiological factors responsible for the endocarditis or the degenerative changes underlying the stenosis have already been so fully considered in the causation of the foregoing valvular defects that it would be a needless repetition to discuss them here.

Symptoms.—Stenosis of an orifice is justly regarded as a serious affection; yet in this particular lesion subjective symptoms are sometimes entirely wanting. Consequently the clinical features of each case, as well as their severity, depend upon the degree of narrowing. If this is extreme, it is impossible for the disease to remain latent, and the patient suffers either from a too inadequate supply of arterial blood to maintain nutrition and normal visceral function, or from the effects of stasis behind the seat of constriction.

In the slighter degrees of aortic stenosis patients are usually capable of ordinary physical and mental activity the same as their companions. I recall a lad of fifteen who presented himself at my clinic in the Post-Graduate Medical School, because of some trivial digestive disorder, and in whom were discovered all the signs of uncomplicated aortic stenosis. Judged by the secondary physical signs it was not very pronounced, and the boy stated that he was a newsboy selling papers on the suburban trains, in the habit of carrying heavy bundles of papers, and of jumping on and off moving trains without any shortness of breath or consciousness of his heart's action.

It is in such cases as this that individuals go for years without knowing there is anything wrong with them, and at length learn of their defect through its accidental discovery by some medical examiner. Indeed, persons with thoroughly compensated aortic obstruction may pass through their entire lives to old age without having ever learned of their disease. The pulse of such an individual is slower and smaller than normal, and the cardiac impulse denotes hypertrophy, but having grown up, so to speak, with these deviations from the general rule, he pays them no attention.

If any circulatory disturbances result from the aortic constriction when single and of minor degree, they are such as indicate deficient supply of arterial blood to the various organs and parts of the body. Even these effects may be too slight to attract special attention. At the most, the circulation is not very active, and the boy may not be quite as vigorous as his healthy play-fellows.

It is stated by some authors that aortic narrowing of considerable degree may occasionally give rise to distinct symptoms of cerebral anæmia—i. e., syncopal attacks and epileptiform seizures. Such serious effects must indicate either an extreme grade of obstruction or periods of cardiac weakness when the left ventricle expels very small amounts of blood, or perhaps none at all, for a few seconds, in consequence of intermittence. A far more common symptom is vertigo; and yet even in this there is nothing peculiar to aortic stenosis, since, as well known, dizziness may be experienced in any form of valve-lesion. In one instance coming under my observation attacks of vertigo proved a most distressing feature, and yet in this case they depended not so much upon the cardiac defect *per se* as upon disordered action of the muscle fibres —i. e., interference with its capacity for conducting motor impulses. The case presents features that bring it into the category of Stokes-Adams disease, and will be referred to again in considering that interesting symptom-complex.

In the fall of 1899 an officer of the United States Army desired my opinion concerning the condition of his heart, which he stated was very unusual. He was highly intelligent, and had made his heart an object of much study. From his detailed report of his history the following summary is given: He was born in 1873, and with exception of measles had no illness until his eighth year, at which time he had a protracted and nearly fatal illness thought to be acute gastritis. For a number of years thereafter his digestion was weak, but he was able to participate in the games and sports of his playmates without being conscious of ill effects.

At the age of fourteen or fifteen he accidentally discovered that his pulse-rate was between 40 and 45, but at the age of eighteen he was passed for life insurance, the examiner finding his pulse of normal frequency, and not detecting any cardiac murmur. Nevertheless, when a year later he applied for admission to the West Point Military Academy he was told that he gave signs of slight aortic stenosis. Thereupon he consulted many physicians in his native town and elsewhere, receiving a variety of opinions. Some declared he had valvular disease, and others as positively asserted the contrary.

On one occasion, after having hopped up and down the examining surgeon's office, he was told that his pulse was beating 140 per minute. To make a long story short, it suffices to say he was at length admitted to the Academy at the age of twenty, notwithstanding the discovery of his aortic stenosis, the lesion being considered trifling and compensation good. During his cadetship he was able to endure the arduous drills with apparently no more distress than did his comrades, although he noticed upon a few occasions that the veins on his forehead stood out prominently.

In his senior year he, like many others of his class, had to go to the hospital with chills and fever that were considered malarial, and which have not recurred since his leaving the Academy. From September, 1897, until the spring of 1899, he was stationed in San Francisco, and while there had a pulse-rate of 38, but with the exception of slight blurring of vision and headache, that was always relieved by calomel, he had no illness or symptoms referable to his heart. In the fall of 1898, upon being examined for promotion, he was told he had aortic stenosis and mitral regurgitation, and was rejected in consequence. Nevertheless, upon his record he at length obtained his promotion, and was transferred to a post in the East.

In May, 1899, he participated in a bicycle ride, being unaccustomed to that particular form of exercise, although he engaged in every other kind of sport and game with his fellows. During this ride he made a spurt, and then got out of breath, but otherwise appeared to suffer no inconvenience. Two hours subsequent to his return to his quarters, and while standing by a table waiting for dinner, he suddenly became dizzy and was assisted to a couch. The junior surgeon was summoned, and finding his pulse 30, administered whisky, which somewhat relieved him and brought his pulse up to 50. From that time on he was daily distressed by spells of vertigo for some weeks, and he noted that the regular slow action of the heart was every now and then exchanged for a more rapid irregular one, with an occasional violent thump against the chest-wall. At this time his dizziness was usually relieved by assuming the recumbent posture.

He received some medicinal treatment, nature unknown, and then for a month was free from his distressing symptom. It returned again, however, more violently and still annoyed him in November, 1899, the date of my first examination. During the summer previous he was given digitalis for a short period, and twice after having taken the remedy his pulse suddenly became accelerated to 60, and was regular. During these months he was in the habit of striving to either work off or forget his dizziness by playing golf, and it is worthy of note that such exercise did not aggravate the symptom.

In September, 1899, he suddenly, while studying the action of his heart, made the discovery that during his spells of vertigo, and while his radial pulse was but 26 to 30, he could perceive by placing his finger above the clavicle a series of " small pulsations which corresponded with feeble heart contractions." These were of variable number, and were interposed between two energetic cardiac contractions which were declared by a forcible apex-impulse and by the radial pulse. He furthermore noted that no matter how slow his radial pulse was, even as infrequent as 19 in the minute, he was not dizzy provided it was regular and the number of small pulsations in the neck was uniform. I shall recur to this striking and peculiar feature again. During that same fall he was treated for a few weeks in the Battlecreek Sanitarium, and while there requested on one occasion that an " ice compress " be placed upon his heart. This was done, and upon its removal ten or fifteen minutes later his heart grew regular and the pulse at the wrist registered 56. He then fell asleep, only to find next morning that its rate was again 26 to 30.

The night before consulting me had been an unusually bad one, and when I saw him the pulse was 36 and irregular. He admitted no shortness of breath on exertion, but suffered from constipation. He thought that on a few occasions he had lost consciousness. Not to make this narrative tedious, I will say my examination revealed hypertrophy of the left ventricle, the apex-beat when felt being broad and strongly thrusting in the sixth left interspace, 11.5 centimetres to the left of the median line and 1 centimetre outside of nipple. Increase of both superficial and deep-seated dulness to the right showed enlargement of the right heart. There was a loud, rough systolic murmur over the entire præcordia whenever the heart made an energetic contraction, and this murmur was succeeded by a distinct second sound even in the aortic area. Upon investigating this bruit minutely it was found to have two areas of maximum intensity, one in the aortic and the other in the mitral area, although I could not detect that the quality in the two regions was different. I found subsequently, however, that at the apex the murmur was softer and more blowing.

The murmur at the base was transmitted upward into the neck as well as on to the body of the heart, while that at the apex was propagated outward into the axilla and feebly to the back. At first I did not observe the small pulsations in the neck, but when my attention was directed to them I perceived them distinctly enough, and I also noted that synchronous with these were feebly audible cardiac tones, while at a much later date these feeble heart-sounds were accompanied by a faint murmur and an indistinct apex-beat. These small pulsations, for lack of a better term, were of irregular number, being ordinarily two, but sometimes as many as seven, and according to the officer even more, before there would come a normal pulse-wave. It was suggested by the patient that these were auricular in origin, but I decided that they were due to feeble ventricular contractions, and subsequent cardiograms taken by Dr. Janeway in New York established the correctness of my opinion.

The very interesting feature pertaining to these incomplete systoles was, that so long as they were regular and in groups of two, vertigo did not ensue. When, on the contrary, they ran up to half a dozen or more the patient felt dizzy or even fainted. These pulsations were first thought by me to be in the carotid artery, but are now known to be jugular.

My diagnosis of the cardiac disease was the same as that of others: aortic stenosis of mild degree and mitral insufficiency, the heart being in a state of still-preserved compensation; for although vertigo was a symptom, there was no dyspnœa on effort and no secondary hepatic or other visceral engorgement. Two things greatly puzzled me: first, if the mitral valve leaked why did not the obstruction of the aortic orifice render the regurgitation through the mitral more serious, as is usually the effect; and, second, what was the cause of the vertigo, or rather the irregularity, in the heart's action.

This latter condition, I believed, was in some manner connected with his digestive organs, but just how I could not decide. The urine was collected for twenty-four hours and carefully examined in the Columbus Medical Laboratory, but aside from some concentration was negative. The genitalia were examined by a competent specialist, but showed nothing more than slight urethral congestion and hyperæsthesia. The patient was sent to an expert neurologist, who was not able to discover any cause in disorder of the nervous system. Diffusible stimulants in frequent large doses for many hours were administered without appreciable effect, as were cathartics and remedies to improve digestion; all to no purpose. The case was dismissed, therefore, as an enigma and without a parallel in my experience. It was not at this time recognised as an instance of Stokes-Adams disease.

But now comes the still more interesting sequel. At Christmastime, 1900, this same young officer reappeared with the statement that during the previous summer he had visited Bad Nauheim and been treated with baths by Dr. Schott without any benefit; had been examined by a number of competent men, among whom was Rosenbach of Berlin. No one had given him any help, and no one had decided the precise nature of his heart-trouble. Rosenbach indeed had diagnosticated the aortic stenosis, but was undecided as to what was the condition at the mitral. I found things exactly as a year earlier. But strong in my belief that the vertigo that still continued was in some way related to his digestion, or metabolism, or excretion, or all together, I persuaded the young man to try for a month an absolutely non-animal dietary, by which was meant the exclusion of anything derived from the animal kingdom, including meat, poultry, fish, eggs, etc., with exception of cheese and milk. These and butter, of course, were to be allowed, together with all kinds of cereals, vegetables, fruits, nuts, and breads. For several weeks his urine was sent to me for analysis, and was always loaded with indican and oxalic acid, but in other respects was normal. Nearly four months later the patient reappeared with a perfectly regular pulse of 26, and was entirely free from his vertigo, which he stated had left so soon as he began the prescribed diet and had not once recurred.

Examination of the heart showed the condition unchanged excepting regularly interposed between every two vigorous cardiac systoles were two feeble contractions that produced palpable and visible small pulsations in the right common carotid, as well as weak heart-sounds, but no perceptible radial pulse. The actual heart-rate was therefore 78. The subjoined sphygmographic tracing (Fig. 65) was taken by Dr. Edward F. Welles, and is by him, a competent judge, considered entirely normal except in rate.

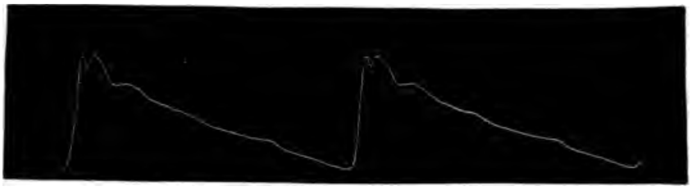


FIG. 65.—SPHYGMOGRAM FROM CASE OF AORTIC STENOSIS (p. 324). Pulse-rate, 25 per minute.

The conclusions to which I am forced by this case are the following: 1. That this singular cardiac action is normal to this individual, and that it is only its irregularity that occasions symptoms of any kind. 2. That this irregularity was of an auto-infectious origin, due either to the production of leucomaines or to the influence of constipation on the vagus or heart-muscle cells, for on this new dietary he has had two or more bowel movements daily. 3. That the lesion is a mild aortic stenosis together with a mitral defect, of the exact nature of which I am not certain. But as I feel sure that at my last examination I detected a very short presystolic thrill and murmur as well as an apex-systolic murmur, I am inclined to the opinion that there is both narrowing and regurgitation at the mitral ostium. Why this combined lesion does not occasion secondary signs and symptoms can only be explained on the hypothesis that the defects are slighter than would be supposed from the cardiac findings, particularly the intensity of the murmurs. The peculiarity of his normal, or at least seemingly normal, cardiac action, is still inexplicable. The symptoms in this case do not as such belong to the usual history of aortic stenosis, but are here narrated because they may be remotely referred to the valvular lesion and illustrate the vertigo said to be sometimes dependent upon narrowing of the aortic ostium.

It is apparent that the development of subjective symptoms is determined by other conditions than the mere existence of aortic stenosis. They depend upon either disordered or deficient cardiac action. When at length, either in consequence of overstrain or of extreme narrowing, the left ventricle begins to manifest inadequacy, dilatation sets in, and it is no longer able to completely empty itself. Symptoms of stasis back of the point of constriction now appear. Left ventricle weakness may be shown by feebler systoles and a more rapid, even irregular pulse. Always small and of low tension, it now becomes still emptier, and at the same time more rapid than formerly.

When in the course of time dilatation leads to relative mitral insufficiency, the clinical features become those of mitral regurgitation in an intense degree. Even before things have reached this grade, however, the patient has noticed more or less breathlessness on exertion, and it may be also attacks of palpitation.

If now the heart is carefully percussed, it is found that the right ventricle has increased in size, while there are in addition signs of engorgement of the general venous system. This condition may last for months before relative mitral insufficiency becomes pronounced, but as a rule the condition grows more or less rapidly worse and the individual is no longer able to keep about because of dyspnœa and congestion. A mitral systolic murmur is added to that of the aortic stenosis, the right ventricle, feeling the strain of pulmonary congestion, begins to pulsate in the epigastrium, the cervical and superficial veins swell, the liver grows palpable and tender, the urine becomes scanty and concentrated, and at last œdema makes its appearance in the feet and ankles. The case has now become converted into one of mitral disease in the stage of broken compensation.

When the mitral orifice shares in the dilatation of the ventricle, the ensuing regurgitation acts as a safety-valve, the same as in cases of aortic incompetence, and actually serves to prolong life. Not infrequently the strain on the right heart leads also to relative tricuspid leakage, and we have the signs of that lesion added to those of the aortic and mitral defects. When this state is reached the patient's sufferings are often extreme, and may be protracted through many months.

In the spring of 1898 I first took charge of a lady of forty-one who had an extreme and apparently pure stenosis of the aortic orifice of rheumatic origin. Six years before, the late Dr. W. W. Jaggard delivered her of a son, and recognised the gravity of the case because of the valvular lesion. The lady did not subsequently experience distinctive cardiac symptoms until the care of a consumptive step-son compelled her to reside for many months at a considerable altitude. Going with him first to Colorado Springs (6,000 feet), she there suffered intensely from shortness of breath, so that they were obliged to take up their residence in the Pecos Valley at a height of about 2,500 feet. Even there she was not able to walk without considerable dyspnœa.

After the death of the young man this lady went abroad, and in Europe sought medical advice. She was advised to take a course of baths under Dr. Schott, at Bad Nauheim, and while there had her first violent attack of angina pectoris. This followed one of her baths. No special benefit was produced by the balneological treatment, and she returned to America. During the winter of 1897 and 1898 she passed through an attack of acute nephritis, but when she consulted me the urine showed no albumin or other abnormal findings. The heart was greatly enlarged, but compensation was still fair. That summer she had a violent attack of angina following a fatiguing walk across a very uneven meadow, and thereafter was never again well.

When I saw her in the fall I considered it necessary to confine her to bed for a number of weeks, and when at length she was permitted to get up she was still obliged to remain on one floor and to move about with great slowness. The action of the heart did not quicken much under exertion, but the pulse grew very feeble, the veins of the neck swelled, and she breathed with evident difficulty. She had several severe anginal seizures, which will be described in the article on Angina Pectoris. Strength was gained with great slowness, and she was rarely free from more or less cardiac distress. This took the form chiefly of breathlessness, distention of the abdomen by flatus, and hepatic congestion.

There was never any œdema, but the ankles often felt swollen and stiff. She passed the summer of 1899 at the seashore under the care of my friend, Dr. Edward O. Otis, of Boston, and in the autumn returned no worse but apparently no better as regarded her heart. All these months she had been kept on approved cardiac tonics and was frequently obliged to resort to hydragogue cathartics because of the relief they afforded. The ensuing winter was a hard one for her, as she was most of the time confined to her apartments in the care of a trained nurse. This was necessary by reason of the possibility of an anginal paroxysm and because at night she would sometimes awake deathly cold, in a drenching sweat, and feeling extremely faint.

The pulse at these times was small and feeble, and the countenance was blue. Prompt stimulation relieved her, but not always speedily, for it seemed as if absorption from the stomach was slow, and hence resort was had to hypodermics of $\frac{1}{100}$ of nitroglycerin, followed at once by heat to the surface and diffusible stimulants. During that winter, and indeed I may say most of the time for nearly three years, the pulse-rate did not vary much from 96, sinking as low as 90 when she was at her best, and when at her worst rising to 105. I believe that on a few occasions I noted a few beats less than 90, usually regular. As a general thing the pulse was tense, and exacerbation of symptoms was invariably preceded by a noticeable increase in its hardness.

This patient was much distressed by frightful dreams, from which she awoke with a start and a feeling of faintness. She was also easily startled by unexpected noises, although she appeared to have her nerves under good control. Insomnia was very distressing, and yielded to nothing so well as to hypnotism. She had to be extremely careful in diet, for at times everything seemed to create gaseous distention of the stomach and bowels with immediate aggravation of her dyspnœa. During that winter relative incompetence of the mitral valve became constant, and every now and then tricuspid regurgitation was added. Even without actual leakage of the tricuspid valve the cervical veins remained much distended and at times caused pain by their pressure. I have since noted painful swelling of the jugulars in another patient, but as a rule have not known patients to complain of actual pain from this cause.

Whenever an attempt was made to invigorate the circulation by considerable doses of digitalis or other cardiac tonics, this patient became annoyed by a feeling in the heart, which she characterized as "pounding," so that the treatment finally settled down to an attempt to keep down pulse tension and stasis by means of nitroglycerin and cathartics, with strychnine and caffeine in small four-hourly doses, and careful feeding.

Thus weeks dragged into months, spring came and passed, the heated term was at hand, with its thunder-storms, for which she possessed an uncontrollable phobia, and she was again sent down to Dr. Otis, at Rye Beach. By fall her condition grew so threatening from cardiac dilatation and visceral congestion that she was not able to return to Chicago until Thanksgiving. When at last she was able to make the journey and reached home I found her in a deplorable state. Both auriculo-ventricular valves were leaking, and the liver was enormously increased in size. The lungs were so congested that she was harassed by a frequent cough, which completely exhausted her, made the face actually purple, and caused her to gasp for breath for many minutes.

The difficult sputum was often bloody, or if not actually sanguineous was made up of thick, tough brownish mucus. The bases of the lungs were dull, and everywhere were copious moist and dry râles. Heroin, strychnine hypodermically, energetic catharsis, and apomorphine in $\frac{1}{8}$-grain doses at last pulled her out of her desperate condition, and by Christmas she was reasonably comfortable. She was obliged to remain in bed, however, or to exchange this for an invalid's chair. Whenever she made this effort her pulse grew weak, the veins distended, and she was unable to speak for a minute or two on account of shortness of breath.

The excitement and fatigue of the holidays nearly used her up. Her cough returned with increased visceral hyperæmia and became so frequent and distressing that it could only be controlled by hypodermic administration of hydrochlorate of heroin $\frac{1}{24}$ of a grain. This, however, after a day or two produced nausea and vomiting, and then I actually feared the strain of emesis would make her heart stop beating altogether. As it was, after each vomiting spell she sank back on her pillows, blue in the face, gasping for breath and too exhausted to speak, while the perspiration simply poured off of her.

At length, however, things mended somewhat, and if not reasonably comfortable, she was at least not miserable. Then albumin and casts appeared in the urine in large amounts, and this patient sufferer began to fail slowly but steadily. By the middle of January it became necessary to resort to stimulating injections of morphine and atropine to keep off the horrible sensation of fainting which took possession of her. Strychnine was increased to the limit of toleration, and in addition hypodermics of a grain of valerianate of caffeine were also given every two hours.

Stasis became so distressing, although œdema was never a very marked feature, that cathartics became a daily necessity. I recognised that the morphine was a two-edged sword, increasing the danger of uræmia and upsetting the stomach, while at the same time affording her relief from positive misery, and therefore it was not withheld. At length, towards the end of February, this boon became so necessary that more than a grain a day was administered. This sufferer's craving for stimulation became most urgent and distressing—so much so that whenever the effect of the stimulants passed off she at once felt a terrible sensation of dying. Of course this could not be kept up for long, and finally, five days before her death, the stomach gave out. Whether owing to the morphine, to the gastric hyperæmia, to irritation of the nerve-centres, or uræmia, I am not able to say, the vomiting became incessant except when she was under the influence of large doses of morphine, as often as every five hours.

There actually seemed to be no circulation at times, as judged by the state of the venous system, and yet that poor heart kept right on, beating 105 times a minute, and for the most part regularly. It seemed as if the end must come at any moment through diastolic arrest of the organ, and yet merciful death was withheld for five weary days. At length, forty hours before the struggle ceased, I stopped all medication, except what morphine was actually required to prevent unnecessary suffering, in the hope that by so doing the end might be hastened. Still that heart went on, although gradually growing weaker and weaker. Twenty hours prior to death she sank into coma—merciful coma—and at five o'clock in the morning the sufferer suddenly gave a little gasp, there came a gush of blood to her lips, and all was over. Death was probably due to pulmonary apoplexy, in consequence of sudden arrest of the left ventricle an instant before that of the right.

No excuses are offered for the detailed narration of this case, since I believe it is highly instructive. Two years and a half elapsed between the time this patient first took to her bed and her death, and during all these thirty months it was one unceasing fight against the inevitable. The original defect at the aortic orifice became converted, so far as symptoms were concerned, into a mitral and tricuspid regurgitation; but with this difference, that the aortic narrowing absolutely precluded all possibility of overcoming the dilatation of the left ventricle and the closing of the mitral valves. Every now and again treatment closed up the tricuspids, but nothing could restore adequate arterial circulation. The more one tries in such cases to force the left ventricle to contract energetically the more is its dilatation likely to be increased.

In this case there was another element that had to be reckoned with—namely, the angina pectoris and the probable degeneration of the myocardium resulting from the cardiac ischæmia that led to the angina. From the start I foresaw the inevitable result, and we only put up as good a fight as we could.

Physical Signs.—*Inspection.*—In most cases of aortic stenosis there is nothing in the patient's appearance to attract attention unless it be some degree of pallor. Cyanosis is not present so long as compensation is preserved, and therefore when observed it is indicative either of some associated lesion or of cardiac inadequacy that has led to stasis. The chief value of inspection lies in the fact that it enables one to detect the location of the apex-beat. This, in consequence of the hypertrophy of the left ventricle, is seen displaced downward and outward, the extent of displacement depending upon the degree of hypertrophy. In thin individuals with broad intercostal spaces there is sometimes a diffused lifting of that portion of the chest-wall, overlying the left ventricle, but this is rarely so pronounced as in aortic regurgitation.

Palpation.—The hand laid upon the præcordia perceives a slow, broad, heaving impulse, and at once receives the impression of a powerfully contracting organ. Palpation is consequently a valuable means of examination by enabling one to judge of the contractile energy of the left ventricle. In corpulent persons the thickness of the thoracic parietes may conceal the real force of the apex-beat, but as a rule feebleness of the impact, even when the apex is displaced, is a token that dilatation of the ventricle is weakening its systoles. Furthermore, in many cases of aortic narrowing careful palpation of the base of the heart detects a thrill or frémissement at some point along the course of the ascending aorta. This is generally in the second right interspace close to the edge of the sternum, but it may be on the breastbone or in the third interspace, immediately adjoining the left sternal border. The intensity of this thrill is variable, but its rhythm is always systolic. It is needless to remind the reader that this vibration is the palpable expression of eddies or currents in the blood-stream after it has passed the point of constriction.

The pulse of aortic stenosis is small and usually weak in consequence of the diminution in the amount of blood ejected from the ventricle. Its size therefore furnishes some indication of the degree of stenosis. So long as the myocardium is healthy and compensatory hypertrophy is maintained the pulse is regular, and in rate is generally somewhat below the normal. Accordingly, an undue acceleration, or an irregularity, or intermittence of the pulse is a sign of weakness. If aortic incompetence is associated the pulse is likely to be modified in accordance with the characters of that lesion. The sphygmographic tracing of aortic stenosis shows rather distinctive characters. The amplitude is not great, the line of ascent is oblique, the summit rounded, the descent gradual, and the secondary waves indistinct. These characters are shown in Fig. 66, which is the copy of a tracing obtained from one of my patients who presented the signs of pure and uncomplicated narrowing of the ostium, there being no diastolic murmur of regurgitation, and the left ventricle hypertrophied with very little dilatation.

FIG. 66.—SPHYGMOGRAM OF UNCOMPLICATED AORTIC STENOSIS.
Personal observation.

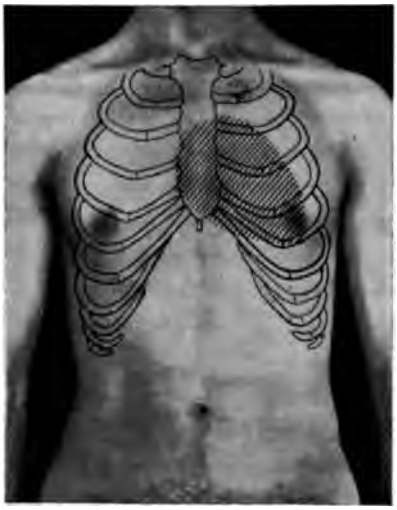


FIG. 67.—TYPICAL RELATIVE DULNESS IN CASE OF WELL-COMPENSATED AORTIC STENOSIS.

Percussion.—So long as compensation is preserved, deep-seated cardiac dulness is increased towards the left and downward to an extent commensurate with the degree of left ventricle hypertrophy (Fig. 67). It is only when failing compensation has led to pulmonary congestion, or when aortic stenosis is united with a mitral defect, that percussion detects any increase of absolute and relative cardiac dulness to the right.

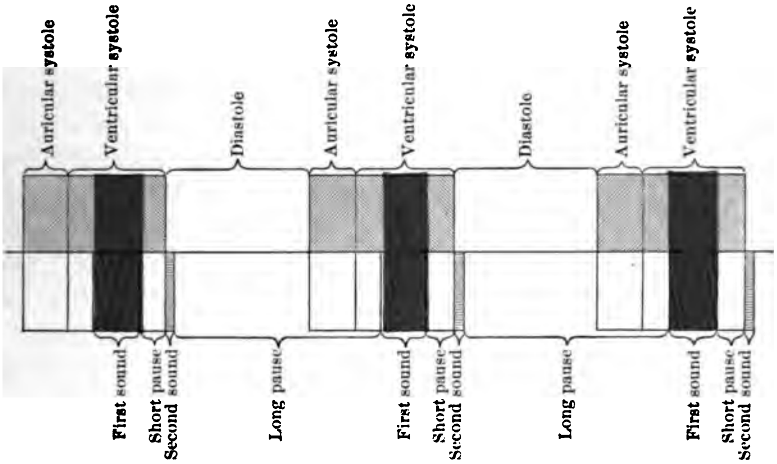


FIG. 68.—RHYTHM OF AORTIC OBSTRUCTIVE MURMUR.

Auscultation.—The first sound at the apex is apt to be dull and muffled in consequence of the preponderance of its muscular element, while the second tone is likely to be enfeebled. Over the base of the heart in the aortic area the ear perceives a murmur which is synchronous with the first sound, and is therefore systolic (Figs. 68 and 69). In pure stenosis there is only this one bruit, but not infrequently there is also a diastolic murmur due to accompanying aortic regurgitation. The systolic murmur, like the thrill, is of variable intensity,

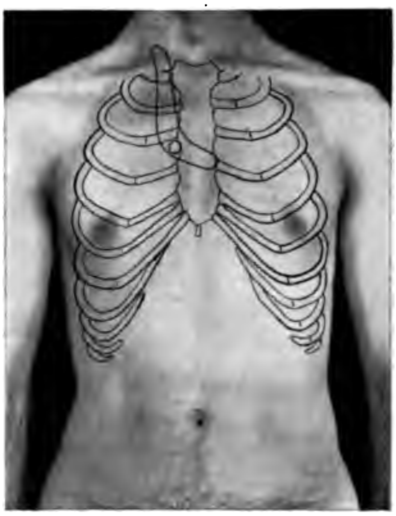


FIG. 69.—PLACE OF MAXIMUM INTENSITY (SMALL CIRCLE) AND PROPAGATION OF AORTIC STENOTIC MURMUR.

but as a rule it is heard with great distinctness, and is of a harsh or grating quality. Its direction of propagation is with the blood-stream upward into the neck, and it is not rare for the bruit to be audible in the left interscapular region along the course of the descending aorta. In exceptional instances when very intense it is heard throughout the præcordia, particularly upon and down the sternum, being sometimes most distinct in the left third interspace over the anatomic seat of the aortic valves. The murmur generally replaces the first tone at the base, and when the valves are too stiff and thick to close, the second tone in the aortic area and in the cervical arteries is wanting or so enfeebled as to be merely a rudimentary click. Consequently, in those cases in which the aortic second sound is retained in its normal intensity and clearness, this fact suggests the possibility that the obstruction is situated in the conus arteriosus or at the ostium, the valves themselves being but slightly affected.

Diagnosis.—As a general thing there is but little difficulty in diagnosticating the disease in question. The conjunction of signs of left ventricle hypertrophy with a systolic murmur in the aortic area and enfeeblement of the second tone at the right of the sternum, is as a rule sufficient evidence for its diagnosis, particularly if the person is under forty, and furnishes a history of a previous attack of rheumatism. There are three conditions, however, that must be differentiated: (1) sclerosis of the aorta or its valves without obstruction, (2) aortic aneurysm, and (3) an accidental murmur, often called anæmic.

In favour of arteriosclerosis are the following: Middle or advanced age, stiffened peripheral arteries, accentuation and ringing quality of the aortic second sound. The left ventricle alone may be hypertrophied, but in most cases the whole heart is enlarged. A history of syphilis as against inflammatory rheumatism also makes strongly for sclerosis instead of stenosis. The difficulty is still further increased by the consideration that degenerative changes may lead to narrowing of the orifice in one way or another. Consequently, a precise differential diagnosis between these two diseases cannot always be made.

As regards an aortic aneurysm—every one knows that when this is small it is often impossible of detection, yet the following differential points may be stated: The patient's age, being forty or more, a history of syphilis or of injury or strain, stiff arteries, symptoms of pressure, as pain, dyspnœa, and cough, inequality in the size of the pulses of the neck and upper extremities, displacement rather than hypertrophy of the heart, pulsation, particularly if expansile and combined with bulging in the aortic area, circumscribed dulness over the manubrium sterni or at either side, and in addition to the systolic aortic murmur, a booming second tone that is not quite pure or is accompanied by a faint bruit. If doubt is still entertained, resort should always be had to the X-ray. Indeed, if this means of diagnosis is accessible, it should be appealed to for confirmation in all cases. Mediastinal tumours pressing on the aorta are so rare that they will not be considered.

If all the signs of aneurysm just mentioned were present in every case a differential diagnosis would not be difficult, but unfortunately such is seldom the case. I recall a patient in the wards of Cook County Hospital who presented a conjunction of signs that rendered the condition of his aortic orifice a subject of much controversy, and owing to his departure from the hospital were never cleared up. In this case there was a history of syncopal attacks, and this fact, it was argued by some, made strongly for stenosis. On the other hand, I felt quite certain of a circumscribed area of deep-seated dulness in the first interspace close to the right sternal margin, and therefore believed the condition was more likely an aneurysm.

Lastly, an accidental murmur in the aortic area may, when occurring in the young, give rise to the suspicion of a stenosis. The error can be avoided by attention to the following points: The absence, it may be, of a rheumatic history, the sex (the patient being most frequently a female, in whom aortic stenosis is comparatively rare), the absence of left ventricle hypertrophy, retention of the aortic second sound, the presence of other accidental murmurs in other areas, as pulmonary and mitral, the softer quality of the murmur, greater frequency of the radial pulse, and the detection of anæmia in some instances.

Prognosis.—This depends upon the etiology of the affection and the degree of compensation that has been established. If stenosis has resulted from degenerative changes in the valves, there are likely to be associated defects in the aortic walls, and it may be in the coronary arteries, which seriously affect the blood-supply to the heart, and hence prognosis is correspondingly unfavourable. Under such conditions compensatory hypertrophy cannot be maintained for long, even if it has been developed. For the same reason there can be but slight hope of its reinstatement after it has once shown indications of breaking. In these unpromising cases the assurance cannot safely be given that sudden death will not take place. In this respect it presents a similarity to aortic regurgitation.

When the stenosis has been produced by endocarditis in the young, the life-prospect stands in direct relation to the degree of narrowing and the perfection of compensation. Cases of moderate severity may exist many years without symptoms, and the patient may be likely to die of some intercurrent disease. This is substantiated by the frequency with which aortic obstruction is discovered post mortem in cases in which its existence was not known or in nowise contributed to the individual's death.

When, however, compensation has once begun to break down, even though the heart-muscle is not greatly degenerated, the prognosis becomes most serious. The loss of compensation is due to initiation or increase of dilatation in consequence of the resistance having become disproportionate to the strength of the ventricle. In most instances, to be sure, the myocardium of the ventricle has suffered degeneration of its contractile elements, in consequence of the small supply of blood sent to the coronary arteries; yet by reason of the progressive nature of the valvular defect the ostium may at length become so reduced in size that even a healthy ventricular wall cannot carry on adequate coronary circulation. In either contingency it is out of the question for the ventricle to again develop predominating and adequate hypertrophy. Accordingly, the prognosis is very different from that of failing compensation in mitral regurgitation, in which, if not too badly lost, it may be repeatedly restored.

Mode and Causes of Death.—Stenosis of the aortic orifice *rarely terminates in sudden death.* Certainly it possesses no inherent tendency to such an end, as aortic regurgitation may be said to possess. In obstruction the failing ventricle tends to gradual, not sudden dilatation, and hence the fatal issue is likely to come through the effects of progressing stasis, the same as in mitral defects. In some cases increasing weakness of the heart ends in fatal exhaustion before œdema and transudation into the serous cavities become marked. The last weeks, or even months, of life may accordingly be highly distressing to both patient and friends, and death be welcomed as a deliverer.

Of 20 cases of aortic stenosis Hustedt found the following causes of death: Cardiac asthenia 2, pulmonary phthisis 7, pneumonia 3, marasmus 2, pulmonary œdema, apoplexy, nephritis, bronchitis, emphysema, carcinoma, and anæmia, each 1. It is noteworthy that but 2, or 0.9 per cent, were attributable directly to the heart, while the remaining 20 were due to intercurrent diseases. It is also interesting to note that in 7 cases death was due to pulmonary tuberculosis, and that therefore aortic stenosis may be said to predispose to this disease, probably in consequence of the general malnutrition, which is favoured by obstruction at the aortic orifice. This finds further corroboration in the fact that 2 died of marasmus and 1 of anæmia.

Summary of Physical Signs of Valve Lesions of the Left Heart

	Apex-beat.	Cardiac dulness	Murmurs.	Secondary signs.	Pulse.
Mitral regurgitation...	Increased in strength and displaced to left.	Increased chiefly downward and to the right, but also to the left.	Systolic at apex and transmitted outward, obscuring or replacing first sound.	Accentuation of pulmonic second sound and enlargement of right ventricle.	Of low tension, accelerated and often irregular.
Mitral stenosis........	Thumping, but slightly, if at all displaced, preceded by thrill.	Increased to right and downward, rarely to the left.	Presystolic, limited to apex, with sometimes an early diastolic.	Thumping first and doubled second sound at apex. Accented pulmonary second tone. Enlargement of right heart.	Small, weak, slow, regular or irregular.
Aortic regurgitation...	Broad, heaving, displaced downward and to the left.	Increased to left and downward.	Diastolic, replacing second sound in aortic area, transmitted downward and to the left.	Enlargement of left ventricle. Systolic femoral snap. Duroziez's sign. Capillary pulse, sometimes venous pulse.	Collapsing.
Aortic stenosis........	Broad, thrusting, displaced to the left.	Increased to the left.	Systolic, replacing first sound in the aortic area, transmitted upward.	Systolic thrill in aortic area, feeble aortic second sound. Hypertrophy of left ventricle.	Slow, small, of low tension, regular.

CHAPTER X

TRICUSPID REGURGITATION

This is the most frequent of the valvular lesions which affect the right heart. It is divisible into two classes: 1. Structural. 2. Relative or functional. By this latter is meant an incompetence of the valves, which is due to stretching of the orifice, and is the more frequently encountered.

Organic disease of these valves is one of the rarer cardiac defects, and when found as a chronic affection is generally congenital. It is not as a clinical entity that tricuspid insufficiency is rare; it is only the structural deformity of inflammatory or sclerotic nature that is rare. Concerning the frequency of relative tricuspid regurgitation Gibson says: "It is incomparably the most common of valvular lesions, and that the reason this fact is not brought out in statistics upon the relative frequency of valve defects is to be found in the circumstance that incompetence of the tricuspid valve does not in itself seriously impair the general course of the circulation, and it is therefore often found among those who, although under treatment for various affections, have no cardiac symptoms. It accordingly escapes observation unless especially sought for."

This is a remarkable statement, and is at wide variance with the opinion generally entertained. It would seem a piece of temerity for me to take issue with Gibson on this point, but as I am not willing to accept the presence of a systolic whiff in the tricuspid area as conclusive evidence of leakage at this orifice, I must conclude that I have overlooked this regurgitation many times when he would have diagnosed it. Doubtless, in consequence of the readiness with which the safety-valve action of this incompetence is brought into play, there may many times be slight leaks that are not considerable enough to produce positive venous pulse in the cervical veins and liver; but this is a matter of conjecture rather than of demonstration, and one might argue that the murmur is due to some other condition than actual regurgitation.

Morbid Anatomy.—The changes discovered at the tricuspid orifice, whether they constitute a structural or a relative defect, are analogous to those at the mitral, and therefore a detailed description of them is omitted. In most cases in which the incompetence is owing to defects in the valve itself there is a combination of both regurgitation and obstruction. Consequently, more will be said on this subject under the head of Tricuspid Stenosis. When organic changes at this ostium are encountered, they are usually associated with lesions at other orifices, chiefly the mitral.

When the valves in question are relatively insufficient the right ventricle is found dilated and its wall thin. The trabeculæ are apt to show evidence of hypertrophy, the papillary muscles of having been stretched, and the valve-flaps are often longer and broader than normal, in consequence of the prolonged pressure to which they have been subjected. The auriculo-ventricular ring is also stretched, admitting more than four fingers. The right auricle is dilated, in some instances to an enormous extent, and its normally thin wall is still thinner. It is not uncommon also to find that the distending force of the regurgitant stream has induced more or less dilatation of the great venous trunks close to their termination in the auricle.

The myocardium of the ventricle and auricle generally furnishes evidence of prolonged stasis in the coronary veins, or of degeneration. Finally, there are the associated changes in the lungs or left heart, which have served as the etiological factors in the development of the right ventricle dilatation and eventual incompetence of the valve.

Tricuspid regurgitation is a pathological condition, and yet Adams, and later Wilkinson King, have pointed out that it really exerts a "safety-valve action." It occurs with remarkable ease, and these authors claim it is a beneficent provision on the part of Nature by which the heart is spared from disastrous overstrain. In the chapter on Aortic Regurgitation I pointed out that relative incompetence of the bicuspid valve acts in the same way. But whereas the firmness of the mitral ring renders its stretching a matter of much difficulty, the tricuspid orifice yields to relatively slight pressure and closes down again so soon as the strain is removed. Consequently, as every clinician knows, leakage through the right auriculo-ventricular valve will come and go many times in the course of any disease that throws excessive strain on the right ventricle.

Etiology.—Structural defects at the tricuspid orifice are generally produced during fœtal life, and are the result of endocarditis. Nevertheless these valves may be the seat of an inflammatory process after birth, as well as, although but seldom, of sclerotic changes, the same as other valves. When endocarditis attacks the right heart it is usually associated with inflammation elsewhere in the heart, at one of the other orifices, and owes its origin to the same etiological factors, which do not require recapitulation here.

I shall therefore pass on at once to the consideration of those diseases and conditions that are responsible for the causation of the *relative* form.

Comprehensively stated, these are all those conditions which raise blood-pressure in the pulmonary system to such a point that the right ventricle is no longer capable of successful resistance. Occasionally this pressure becomes so high as to lead to relative incompetence of the pulmonary valves also, but in most cases the ring into which they are inserted proves equal to the strain, so that it is the ventricular wall and basal ring of the tricuspid valve which give way. This degree of abnormal blood-pressure is most frequently presented in mitral disease, particularly stenosis, and hence it is in these cases when compensation is wholly destroyed that relative tricuspid regurgitation is most frequently recognised. Oftentimes it follows the mitral incompetence secondary to dilatation of the left ventricle in cases of aortic valvular disease, and it is very frequently seen in the terminal stage of chronic nephritis.

In renal cirrhosis, in particular, blood-pressure is high and sustained, throwing great strain on the left ventricle. In time this chamber, because of degeneration or of the excessive peripheral resistance, begins to yield, dilatation supersedes the hypertrophy, undue pressure is thrown back upon the right heart, and the tricuspid begins to leak. Thus, whatever is the nature of the primary cardiac disease, the ultimate effect is the same—namely, augmentation of blood-pressure in the pulmonic vessels and right ventricle until a point is reached at which the wall of the ventricle and the auriculo-ventricular ring must stretch.

Other diseases that produce the same effect are vesicular emphysema with or without chronic bronchitis, long-standing bronchial asthma, cirrhosis of the whole or even of a part of one lung, fibroid phthisis, and pulmonary collapse in consequence of pleuritic effusion. A hydrothorax, itself consecutive to inadequacy of the heart, may by compression of the lung hasten or intensify the effect on the right ventricle of primary disease of the left heart. No doubt in some of the pulmonary affections the strain on the right ventricle is intensified by frequent and severe fits of coughing. It is probably in this way largely that tricuspid regurgitation is produced in cases of chronic bronchitis, although many times there is an associated emphysema.

The wall of the right ventricle is thinner than that of the left, and one can readily understand that less internal pressure is required to bring about overdilatation and relative tricuspid incompetence; and yet I cannot refrain from expressing wonder at what Gibson says concerning the influence of fever and other conditions in the production of this valvular insufficiency. "Pyrexia, if of more than brief duration, almost invariably leads to dilatation of the right ventricle and tricuspid regurgitation. It does so sometimes from simple relaxation of the muscular substance, but in other cases by means of hyaline degeneration. Toxic influences belonging to almost every class produce the same effect; the toxines produced by micro-organisms (sometimes in the absence of all pyrexia), the organic poisons, such as alcohol, the inorganic poisons, such as lead, act in precisely analogous fashion. Malnutrition, whether arising from some morbid process, as malignant invasion, from deficient absorption, as in such a simple affection as dilatation of the stomach, or from some deficiency of the food, all lead to the same end. A long experience of out-patient service in our great hospitals enables me to bear witness to the extreme frequency of tricuspid regurgitation in atonic conditions of the stomach. Such disorders as anæmia, in which the nutritive power of the blood is lowered, are also to be considered as potent causes of tricuspid regurgitation."

Severe muscular exertion, as mountain-climbing, may and not infrequently does produce dilatation of the right ventricle and the safety-valve action of tricuspid incompetence. In such instances the protective action of this leak comes beautifully into play, for did the valve not give way and allow the strain to fall on the right auricle, great veins, and liver, the continuance of the exertion would eventually lead to dangerous hæmoptysis or fatal cardiac syncope from overdistention of the ventricles.

I have within the past twelve months seen two stalwart football players who, judging from the history and the subsequent condition in which I found their right heart, must have gotten up tricuspid regurgitation during a game. In both, the ventricle and cervical veins still showed permanent ill effect of their violent exertions. So long as the myocardium of robust young men is healthy, ultimate recovery is the rule; but when after middle age myocardial degeneration exists, individuals should beware of physical efforts that are likely to so seriously overstrain their hearts.

Symptoms.—As a matter of fact tricuspid regurgitation exists so rarely alone—that is, independently of some other cardiac or pulmonary disease—that our knowledge of its symptomatology is in reality derived from our observation of the effects it produces in conjunction with such disorders, or with tricuspid stenosis. Nevertheless, it would not be difficult to deduce the symptoms from our knowledge of the influence of this affection on the circulation.

The first effect of the regurgitation is to hinder the free flow of blood out of the right auricle, and thus bring about its dilatation. This reacts upon the contents of the two venæ cavæ, raising pressure within them from a negative to a positive one. As negative blood-pressure within these two great venous trunks is necessary to the maintenance of the circulation, a rise of blood-pressure within them and their tributary veins tends to bring the bloodstream to a standstill. Stasis thus induced shows itself by cyanosis and turgescence of the superficial veins of the upper and lower extremities and by passive engorgement of the abdominal viscera. The liver grows large and tense, even to the extent of tenderness and pain, particularly in the epigastrium. Functional visceral disorders in various form show themselves, the feet and ankles swell and ultimately become œdematous.

The patient is weak and easily fatigued, and after a time is obliged to keep to his room or even to his bed. Dropsy increases, and may invade the entire body, or ascites and hydrothorax may predominate over the anasarca. Indeed, Gibson, who appears to have had a remarkably rich experience in this class of cases, says: "The fact must never be overlooked that right-sided disturbances are more likely to produce interference with the functions of the pleura than affections confined to the left side of the heart, inasmuch as the blood circulating in the pleural membrane is in overwhelming proportion returned to the heart by the bronchial veins, which discharge their contents on the right side by means of the vena azygos, and on the left side by means of the superior intercostal veins. Their destination is therefore the right auricle. When disturbance of the function of the right heart occurs, there is as a consequence considerable liability to backward pressure upon the pleural membrane, resulting in hydrothorax."

If the tricuspid regurgitation is not secondary to heart or lung disease, dyspnœa and cough are likely to come on only after the growing stasis in the venous system has, through its effect on the general capillary and arterial circulation, led to pulmonary congestion. In most cases, however, these symptoms are complained of prior to the development of the tricuspid leakage, because forming a part of the symptomatology of those diseases to which the tricuspid defect is usually secondary. Therefore, as a matter of fact, the symptomatology of this affection is inseparably linked with that of the antecedent disorders, and does not require recapitulation. According to Gibson, it is possible for tricuspid regurgitation to exist without producing any symptoms, and this is one of the reasons why it is so frequently overlooked. For my part I cannot see how this can well be, and I am not convinced by his statement, for although tricuspid insufficiency may not produce cardiac symptoms, strictly speaking, such as dyspnœa on exertion, still it cannot fail to exert decided effect on the venous circulation in general, which would be sufficiently serious to bring the patient to a physician.

When this valvular incompetence arises in consequence of long-standing heart or lung disease, it speedily aggravates the pre-existing symptoms. The rapid appearance of general dropsy which usually follows the establishment of regurgitation is due in large part no doubt to interference with the lymphatic circulation. The great veins into whose blood-stream the contents of the thoracic duct are emptied are so turgid that the stasis retards the ready emptying of the duct. Congestion results, therefore, in the duct and its tributaries, injuriously affecting nutrition and increasing the permeability of the capillary walls. This disturbance of the circulation inevitably results from primary tricuspid regurgitation, and hence I cannot conceive of the disease remaining latent.

Physical Signs.—*Inspection.*—Contrary to what is usually the case in diseases of the heart, inspection and palpation afford the most, and according to some the only, reliable means of diagnosis in tricuspid regurgitation. This is partly due to association of this lesion with other cardiac or pulmonary diseases that produce conflicting physical signs, and partly to close anatomical connection between the right chambers of the heart and the great venous trunks, in consequence of which the contents of the latter are directly exposed to pressure by the reflux stream in the manner already described. Instead of the large veins which enter the thorax being invisible, the dilatation of the right heart leads to their permanent turgescence, and in extreme degrees even to dilatation of the venous bulbs at the root of the neck. This turgidity is specially marked, therefore, in the jugulars, which may stand forth like great purple cords.

When incompetence of the tricuspid valve permits the ventricular systole to drive part of its blood back into the auricle, a nearly synchronous wave is transmitted upward into the veins of the neck through the superior vena cava and downward through the inferior vena cava, even to the liver or beyond. This reflux venous wave declares itself in the neck as a visible and even palpable pulsation. This is particularly pronounced in the right internal or external jugular, or in both. This phenomenon has been carefully investigated by Riegel, and by him shown to coincide with pulsation in the arteries, as the carotids. This "positive venous pulse," as Riegel calls it, is usually spoken of as systolic, but is, strictly speaking, presystolic-systolic, and may be correctly timed by comparison with the carotid pulse. Simultaneous palpation of the artery and inspection of the vein will show that pulsation in the latter takes place during the rise of the arterial pulse.

In cases in which there is dilatation of the auricle, yet without tricuspid regurgitation, a venous pulsation may likewise be detected, but its rhythm agrees with the last portion of ventricular diastole, including, of course, auricular systole, and hence is, strictly speaking, diastolic-presystolic. This venous pulsation occurs, therefore, during the collapse of the carotid artery. This diastolic-presystolic or " negative venous pulse," as it is called, never indicates tricuspid insufficiency.

The positive jugular pulse of tricuspid incompetence must also be distinguished from a pulsation sometimes communicated to the distended vein from the adjacent artery. This can be done by pushing the artery away from the contiguous vein in the case of the internal jugular. To test the external jugular it should be compressed by the finger a short distance above the clavicle, when, if the pulsation is communicated from an adjacent artery, the part below the point of constriction will entirely or partially collapse and the pulsation disappear wholly or in part.

Palpation.—The corresponding positive venous pulse conducted downward to the liver is to be detected by palpation of the organ. If the congested liver is grasped by the two hands, the left pressing it strongly upward from behind and the right being outspread upon the organ in front, pulsation of the liver is perceived as an expansile distention in all directions. This positive venous pulse in the liver is therefore quite unlike the merely rising and falling motion imparted to it by the pulsations of the abdominal aorta or the downward impulse of the hypertrophied right ventricle above.

This positive venous pulse is the pathognomonic sign of tricuspid regurgitation. Without it, in either the cervical veins or the liver, a diagnosis of this valvular lesion is always open to doubt. There are two exceptions, but these occur so rarely that they seldom need to be considered. One is a wave communicated by the mitral regurgitant stream through an open foramen ovale to the contents of the right auricle, and thus to the stream in the jugulars. The other is a systolic pulsation in the jugular veins due to the rupture of an aortic aneurysm into the vein, instances of which accident may be found in the literature.

The radial pulse presents nothing distinctive even in primary tricuspid incompetence without other cardiac disease. It is small and weak, accelerated and regular, or irregular and intermittent, as the case may be. Popoff has reported diminution in the size of the right radial as compared with the left, and attributed it to pressure of the distended right auricle and veins upon the right subclavian artery.

Dropsy is usually present in cases of relative insufficiency of the tricuspid valve, having in most instances begun before the valve gave way. It is not present, however, in all cases, certainly in the early stage of the tricuspid leakage. Consequently this absence of cutaneous œdema, notwithstanding great venous stasis, is a proof that something more than stasis alone is necessary for the production of dropsy. This additional factor is, as previously stated, an abnormal permeability of the capillary walls depending upon defective nutrition.

Percussion.—This yields information of minor diagnostic importance, because any alteration discovered in the area of cardiac dulness may be due to an associated or antecedent cardiac or pulmonary affection. Vesicular emphysema, chronic pleuritic effusion or hydrothorax, and cirrhosis of the right lung, may render unavailing any attempt to determine by percussion the accurate size of the right heart. In vesicular emphysema the borders of the lungs are distended, pushing the heart away from the chest-wall and occasioning such a degree of hyperresonance that the limits of deep-seated cardiac dulness become inappreciable. When fluid exists in the right pleural cavity, or there is solidification of the right lung, the dulness thus occasioned blends indistinguishably with that of the heart. Under favourable conditions, however, cardiac dulness is found increased to the right and downward, the extent of this increase being determined by the degree of the dilatation of the right ventricle. In cases of primary or independent tricuspid insufficiency due to endocarditis, the right ventricle is found less enlarged

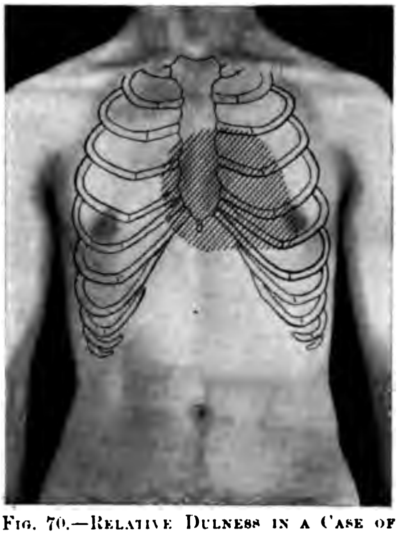


FIG. 70.—RELATIVE DULNESS IN A CASE OF PRIMARY TRICUSPID REGURGITATION.

(Fig. 70) than when regurgitation takes place as a result of ventricular dilatation (Fig. 71). On the other hand, in either form of the affection, dulness is greatly increased over the right auricle and the large venous trunks, reaching far beyond the right sternal border, half-way or more to the right mamillary line. In most instances also there is increase of cardiac dulness to the left, depending upon the nature and extent of the accompanying disease of the left heart.

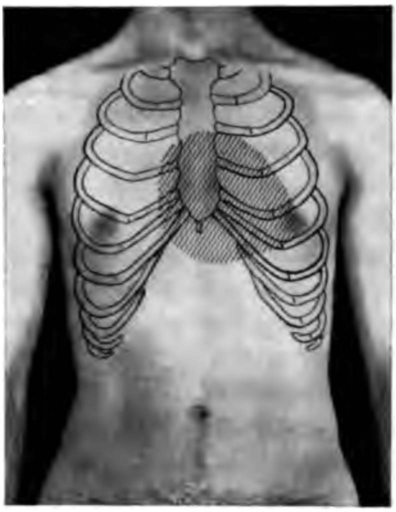


FIG. 71.—RELATIVE DULNESS IN CASE OF TRICUSPID REGURGITATION, SECONDARY TO DILATATION OF THE RIGHT VENTRICLE.

Auscultation.—This furnishes even less trustworthy data than are obtained by percussion. There is generally a blowing, systolic murmur, said to have its maximum intensity in the tricuspid area (Fig. 72); yet as the dilatation of the several cardiac chambers alters the normal relations of the parts, this murmur may be heard most distinctly in any one of several situations. It may be at the junction of the fifth and sixth left costal cartilages with the sternum, over the ensiform appendix, or even to the right of the sternum in the third, fourth, or fifth intercosal spaces, close to this bone.

Gibson, in his remarks on the Heart in Debility, has narrated cases showing that the murmur may be heard in the second left interspace an inch from the sternum, in an

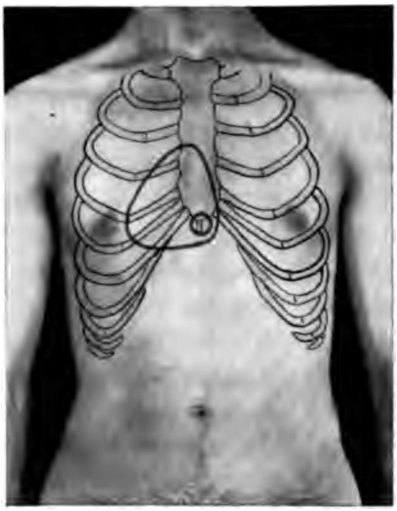


FIG. 72.—PLACE OF MAXIMUM AUDIBILITY (SMALL CIRCLE) AND AREA OF PROPAGATION OF TRICUSPID REGURGITANT MURMUR.

area in which a systolic pulsation is also often observed. In these cases there was also venous pulsation in the neck, and hence it seems probable that the murmur was that of the disease now under discussion. This is the site of a systolic murmur frequently audible in chlorosis and anæmia, and variously explained by Naunyn, Balfour, Russell, Bramwell, Handford, Foxwell, etc. (see introductory chapter), and therefore caution is required in the correct interpretation of a bruit in this situation.

The tricuspid murmur has a blowing quality, is of no constant pitch, and differs much in loudness, according to the conditions that generate it. It is often obscured by other bruits originating at other orifices, particularly at the mitral. If the auscultator is experienced, and conditions are favourable, he may be able to locate different areas of maximum intensity for the different murmurs, and thus be able to determine which is tricuspid, which mitral, etc.

Regarding the heart-sounds in tricuspid insufficiency but little need be said. The first tone over the right ventricle is apt to be muffled, even replaced, by the murmur. The pulmonic second sound appears to differ in different cases. It would naturally be enfeebled, in consequence of the fact that lessened blood is expelled into the artery, but as the predisposing mitral or other disease has augmented blood-pressure in the vessels of the lungs, the second tone in the pulmonic area may be accentuated. However, if in a given case of gastrectasis tricuspid regurgitation is suspected, an enfeeblement of this second sound would lend a measure of support to the diagnosis.

Lastly, it is quite common to hear a vascular tone, if one auscultates the vein in which the positive pulse is seen, and the tone thus obtained is, of course, synchronous with the pulsation.

Diagnosis.—Recapitulating, I wish to emphasize the statement that inasmuch as mitral murmurs may sometimes be heard with great intensity over the right ventricle, and be conducted far beyond the right border of the sternum, it is very unsafe to rely upon a murmur in the tricuspid area in making a diagnosis of regurgitation through this valve. Certainly it is so exceptional for any considerable leakage to occur at this ostium without giving rise to the venous and hepatic pulsation already described, that in the absence of these pathognostic signs it is unsafe to declare that the murmur is that of regurgitation. This has been impressed upon me many times.

There is a certain Russian Jew who exhibits himself to medical students for examination because of his possessing a musical murmur of obscure origin. In his instance the bruit is systolic, and most intense upon and immediately roundabout the xiphoid cartilage. From its location, therefore, it is thought by many good observers to be a tricuspid regurgitant one. The musical murmur is, however, also distinctly audible well outside the left nipple; and as there is a combination of lesions in this case it is very difficult, if not impossible, to definitely decide whether the murmur in question is tricuspid or mitral. It all depends on the existence or not of a positive venous pulse in the jugulars. Six years ago I did not detect such a pulsation; two years ago I thought such a venous pulse was present; a few days ago (March, 1902) there was no such evidence of tricuspid regurgitation, and consequently I am obliged to still leave the question *sub judice*. The heart was in a far better state than two years before, and it is quite possible that a slight relative tricuspid incompetence was accountable for the jugular pulsation at that time. At all events this interesting case is very exceptional, for ordinarily it is not a difficult matter to determine the existence of tricuspid leakage. It illustrates that in the absence of a positive venous pulse in jugulars or liver one is not wise in declaring a systolic bruit in the tricuspid area to be tricuspid.

Prognosis.—This may be said to depend upon the nature and causes of the tricuspid insufficiency. Relative incompetence of this valve may come and go quickly, but unless its cause can be removed its tendency is steadily downward, although death may not occur for weeks or even months.

Mode and Causes of Death.—The fatal termination gradually results from either general or cardiac exhaustion, in consequence largely of malnutrition, or from pressure-effects of the dropsy, or from pulmonary œdema, or from some other terminal manifestation of the primary cardiac or lung affection. In other words, there is no mode of death peculiar to tricuspid insufficiency *per se*. In 3 cases of this disease Hustedt found as the cause of death cardiac weakness once, phthisis once, and anæmia once.

CHAPTER XI

TRICUSPID STENOSIS

THIS is the counterpart of mitral stenosis, but is infinitely more rare. Indeed, it is said to be the rarest of all valvular defects—so much so that some writers speak of it merely as a pathological curiosity, and devote very little space to its consideration. It probably occurs oftener than it is recognised, and yet its extreme infrequency may be judged of by the fact that, although many thousand necropsies are annually made, only 154 cases had been recorded in medical literature up to the fall of 1896. Of these, 114 collected by Leudet occurred prior to 1888, while in the next eight years Herrick collected 40 more. Three of these were his own cases, and the total number was brought up to 154. It is to the latter's monograph that I am indebted for much that will be said in the following pages.

Morbid Anatomy.—This differs according to the group into which the respective case falls, for tricuspid stenosis may be either congenital or acquired. The former class is again subdivided into those due to intra-uterine endocarditis and those resulting from some defect of development. In the congenital form there are the usual associated abnormalities, such as stenosis of the pulmonary artery, defective closure of the interventricular sæptum, and patency of the foramen ovale and ductus arteriosus.

When acquired as a result of endocarditis tricuspid stenosis presents changes analogous to those at the left auriculo-ventricular orifice, thickening, rigidity, and adhesion of the flaps. Vegetations may also be found on their auricular aspect, and the neighbouring endocardium is apt to present the grayish-white appearance and thickening characteristic of mural endocarditis.

The shape and size of the opening at the extremity of the cusps are variable, the same as in mitral stenosis. The conditions are also such as occasion incompetence as well as obstruction. The tendinous cords and papillary muscles may in some cases also show the changes of endocarditis. In the great majority of cases tricuspid stenosis is associated with other valvular diseases, as shown in the annexed tables taken from Leudet and Herrick:

Leudet

Tricuspid stenosis alone	11
Tricuspid stenosis with mitral stenosis	78
Tricuspid stenosis, mitral, and aortic stenosis	21
Tricuspid stenosis and pulmonary stenosis	3
Tricuspid stenosis, mitral stenosis, and pulmonary stenosis	1

Herrick

Tricuspid stenosis	1
Tricuspid and mitral stenosis	18
Tricuspid and pulmonary stenosis	0
Tricuspid, mitral, and aortic stenosis	18
Tricuspid, mitral, and pulmonary stenosis	1
Tricuspid, mitral, aortic, and pulmonary stenosis	1
Tricuspid stenosis and endocardium of the left auricle	1
Tricuspid stenosis and aortic stenosis	0

The changes observed in the walls and cavities of the heart are in part secondary to the tricuspid stenosis, and in part to the coexisting lesions of other orifices and valves. The right ventricle usually exhibits combined hypertrophy and dilatation, in consequence largely of the conjoined mitral defect, but if the tricuspid obstruction is great, with but little if any regurgitation, the ventricle is diminished rather than enlarged in size. The chamber upon which the stenosis chiefly reacts is the right auricle, and hence this is more or less hypertrophied and dilated, according to the degree of the stenosis. It has been known to reach a size of two or three times the normal, but because of the thinness of its wall the right auricle rarely undergoes much compensatory hypertrophy. The degeneration of the myocardium is such as is often found in other valvular diseases, and in one of Herrick's cases the right ventricle was covered by a thick layer of subpericardial fat.

Etiology.—Cases of this disease originating after birth are due to endocarditis, and as in other forms of valvular defect of this origin, articular rheumatism appears to be its chief exciting cause. Herrick states that of the 154 collected cases, 30 per cent gave a history of antecedent rheumatism. He also says that syphilis has been assigned as a cause, and that Leudet regards the puerperium as also an etiological factor. This latter fact may possibly have a bearing on the far greater frequency of tricuspid stenosis in the female than in the male sex. The disproportion of the two sexes in this disease is far too patent to be merely accidental, Gibson stating that of 146 cases of tricuspid obstruction, 114 occurred in females and 32 in males.

This disease also occurs most frequently in the early decades of life, the majority of cases falling between the twentieth and thirtieth years. In this respect it is not peculiar, for, as we know, valvular diseases of rheumatic origin are much more frequent in the young than in persons of middle or advanced age.

Symptoms.—The fact that the ablest and most experienced clinical observers have failed to recognise the existence of tricuspid stenosis during life, and that in most cases it has first been detected on the autopsy table, may be regarded as proof that there is no symptomatology peculiar to this affection. Its clinical manifestations, even when such exist, are, moreover, apt to be obscured by those belonging to the associated lesions. Thus, although obstruction at the tricuspid orifice leads to stasis in the systemic veins, the liver, and other abdominal organs, mitral disease does the same; and as the physical signs of this latter affection generally mask those of the tricuspid defect, the symptoms are quite likely to be attributed to the disease in the left heart.

Tricuspid stenosis tends to limit the amount of blood sent to the lungs and left auricle, and therefore there is nothing in this disease tending to produce dyspnœa of effort and other symptoms referable to pulmonary congestion. In fact, were the tricuspid lesion to exist alone, the effect on the lungs would, like that of pulmonary stenosis, be one of anæmia with its tendency to tuberculosis; and it is here worthy of note that in the single instance in which Hustedt ascertained the cause of death, this was "phthisis." It is plain, therefore, that when we leave out of consideration the pulmonary symptoms due to conjoined mitral disease, we must seek the clinical features of tricuspid stenosis in all those perversions of function incident to visceral hyperæmia and in the effects and manifestations of general venous engorgement.

The jugulars are distended, and when hypertrophy of the right auricle is marked, these veins are likely to exihibit a negative—that is, diastolic-presystolic pulsation. This was the case in one of my patients in whom physical signs led me to suspect the existence of tricuspid narrowing, but in which case, unfortunately, a post-mortem examination could not be obtained. The liver bears the main brunt of the secondary stasis, and is consequently greatly enlarged, perhaps tender, and there is furthermore a growing tendency to ascites and œdema.

I have had the good fortune to observe a patient in whom during life there were the classical signs of both mitral and tricuspid stenosis, as will be subsequently described, and whose heart presented such interesting post-mortem findings that the case will be here introduced.

The patient was a Polish Jew under the care of Dr. Kaczorowski, by whom he was brought to me. His age was given as forty-three, and for six years he had been unable to work on account of shortness of breath. There was a history of scarlatina in childhood and of articular rheumatism fifteen years before I saw him, but further particulars were too vague to be trustworthy.

His symptoms were dyspnœa on exertion and a cough, which had existed for six weeks. The liver was engorged and palpable for a distance of 3 inches below the costal arch, and there was distention of the external jugulars.

The heart was greatly enlarged in all diameters, and there were two separate and distinct presystolic murmurs, one at the apex and the other close to the xiphoid cartilage. The diagnosis of a double stenosis was made, tricuspid as well as mitral, and the patient was not lost sight of, although only seen twice in the subsequent sixteen months, the last occasion being five weeks prior to death. Some four months before the fatal issue he began to have œdema of the lower extremities, and to suffer much from difficulty of breathing, amounting to orthopnœa. The anasarca increased rapidly and soon invaded the scrotum, which became so distended that he was compelled to let it hang through a large opening cut for the purpose in the seat of his chair. Cathartics afforded but slight and transient relief, and upon receiving an urgent request from the attending physician to suggest some means of lessening the painful scrotal distention I advised tapping, although it was realized that this would ameliorate the condition for only a short time.

When at length I found time to visit the poor fellow his plight was truly pitiable. He was in a chair, which he had scarcely quitted for many weeks, and he presented signs of moderate ascites as well as extensive œdema of the lower extremities. The scrotum was as large as a child's head, of a purplish-red colour, very hard to pressure, and bathed in bloody serum, which trickled drop by drop into a basin underneath his chair. Owing to the inability of the patient to move or sit in any other position examination was difficult. But so far as it was possible to determine, the cardiac findings were essentially as found and recorded fifteen months earlier.

The pulse was weak, moderately accelerated, and irregular. As cathartics, diuretics, and digitalis had all been used freely, and proved of very little efficacy, no additional suggestions could be offered, and the poor sufferer was reluctantly left to wear out his few remaining weeks of life as best he might. The blessed release came about five weeks later, and Dr. W. A. Evans made the autopsy.

There was much subcutaneous œdema and gangrene of the penis, scrotum, and one of the large toes. The peritonæum and right pleural cavity contained clear serum. The various organs showed the usual changes of long-standing congestion. The heart was enormously enlarged on both sides and was hardened by Kaiserling's solution without being opened (Plate III). However, an attempt was made to discover the condition of the tricuspid orifice by passing the fingers through the great venous opening in the right auricle. The tricuspid ostium admitted three fingers to the second joint instead of four, as is normally the case, and it felt firm and resisting. Moreover, the valve could be felt projecting across the opening.

When at length the specimen had become hardened and it was opened an interesting combination of lesions was presented. The mitral orifice was a mere buttonhole slit, and the endocardium of the left auricle showed the whitish, thickened appearance denoting prolonged high blood-pressure, and its wall was hypertrophied. The valve projected like a cone into the cavity of the ventricle, which was both hypertrophied and dilated. The cords were fused and presented unmistakable evidence of old endocarditis. There was some thickening of the aortic cusps, which had caused moderate obstruction at that orifice, a conclusion justified by the enlargement instead of atrophy of the left ventricle usually found in mitral stenosis. Yet during life this aortic lesion had not produced recognisable signs aside from enfeeblement of the aortic second sound, which had been attributed to the smallness of the bloodstream ejected into the aorta by reason of the mitral disease.

The pulmonary artery presented a remarkably extensive atheroma, due evidently to the long-standing and extreme pressure to which this vessel had been subjected. The pulmonary orifice was relatively dilated, and the valves were of increased size. Consequently, the conclusion seemed warranted that relative insufficiency of this valve had existed and had contributed to the enormous dilatation of the right ventricle. This very great hypertrophy and dilatation had been recognised during life, but had been put down as secondary to the extreme mitral obstruction. The pulmonary regurgitation either failed to produce a diastolic murmur, or it had been overlooked. The pulmonic second tone was noted as feeble, but this was thought due to the diminished amount of blood sent through the tricuspid ring. I feel quite certain that an audible diastolic murmur did not exist.

The right auricle was not only strikingly dilated, but its wall was thickened, and its lining membrane also showed by its appearance to what a high degree of pressure this chamber had been subjected.

The tricuspid ring was rigid, barely admitting the tips of four fingers. The valve leaflets were very considerably thickened and partially united, so that the opening was considerably smaller than the actual tricuspid ring. It was the thick and rigid edge of the posterior flap that was felt in the preliminary examination.

This interesting specimen was submitted to Dr. Gustav Fütterer, and at first he was inclined to doubt the existence of an appreciable tricuspid stenosis. Yet, after a careful examination of the right heart, he arrived at the opinion that in consideration of the marked dilatation of both the right ventricle and auricle and the disproportionate smallness of the tricuspid ring, together with its firmness and thickening, and the condition of its flaps, one could not escape the conclusion that actual stenosis of the ring had existed.

Moreover, the endocardium of the ventricle displayed slight evidence of previous inflammation, probably the same process that had thickened the edges of the tricuspid valve. Nevertheless, it was Dr. Fütterer's opinion that the symptoms had been caused more by the mitral narrowing and the relative pulmonary regurgitation than by the stenosis of the tricuspid ostium. However this may be, I can only assert that it had been sufficient to occasion very positive clinical signs, else I should not have suspected and diagnosticated so rare a lesion in the presence of a pronounced mitral disease to which one might very naturally have attributed the symptoms of stasis. I must therefore stand by my belief that to the tricuspid stenosis is to be attributed a not unimportant share in the production of the unusual degree of general venous stasis as compared with the pulmonic. The right-sided hydrothorax, discovered post mortem, furnished proof of the enormous stasis that had been present in the cavity of the right auricle.

At all events this case illustrates the influence of right heart lesions in the causation of general venous and visceral stasis, while the gangrene bore witness to the profound emptiness of the aortic system.

Physical Signs.—*Inspection.*—A perusal of Herrick's collected cases convinces one that there is nothing in the appearance of these patients to distinguish them from those with mitral disease in the last stages of broken compensation. In Case 27 of his series venous pulse was noticed, but ordinarily there is nothing more than the ocular evidence of venous and capillary stasis.

Palpation.—The pulse is small and weak, and may be regular or irregular, and moderately or greatly accelerated. In Broadbent's case (No. 25 of Herrick's series) the pulse was reported as 100, small, and irregular, while in E[illegible] Smith's case (No. 29) it was recorded as only 60 and small.

[illegible]

There is nothing in these statements that might not also apply to the pulse in mitral disease. [illegible] ally negative so far as the tricuspid lesion is concerned, but in some instances there may be a short, thumping impulse in the epigastrium similar to but distinct from that of the associated mitral stenosis. This was pronounced in the case I have narrated. There was also a short presystolic thrill in the sulcus between the ensiform appendix and the left costal cartilages (Fig. 73), which was plainly shorter and less distinct than that felt at the apex. Between these two there was a space in which no presystolic thrill could be detected, and it was this fact that first riveted my attention. This short thrill ran up to and ended abruptly with the thumping systolic shock mentioned.

It is conceivable that, owing to the heart lying nearer to the median line than usual, a presystolic thrill and sharp systolic shock of mitral stenosis might be felt in the tricuspid area. Consequently, the recognition of these signs in this area alone would not be so suspicious as was the detection, in my case, of these palpatory phenomena in two separate and distinct situations.

Percussion.—Cardiac dulness is increased over the right auricle—that is, at the right of the sternum—but this is not distinctive, since it occurs likewise in mitral disease (Fig. 74). In tricuspid stenosis it is likely to be particularly well marked. It may be said, therefore, that the evidence derived by percussion is valuable, but not positive.

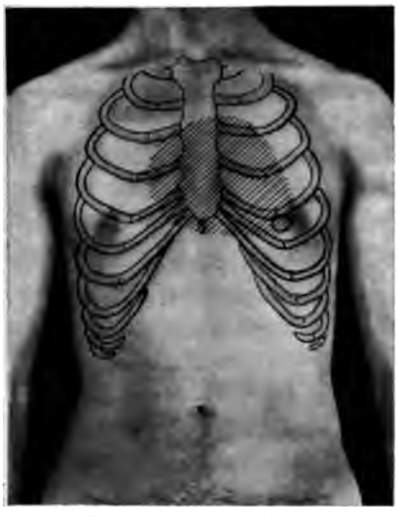


FIG. 74.—RELATIVE CARDIAC DULNESS IN A TYPICAL CASE OF TRICUSPID STENOSIS.

Auscultation. — Unfortunately the results of this means of examination are also likely to be very indefinite. Even if a murmur generated at the tricuspid orifice exists, it is likely to be confused with or indistinguishable from murmurs produced elsewhere, particularly mitral bruits. In my case, as in Broadbent's (No. 15 of Herrick's series), there was a distinctive murmur in the tricuspid area. In my patient a presystolic murmur existed in the very situation in which the thrill was detected, and it was much shorter than that at the mitral area, was of a somewhat different pitch, and terminated in a sharp thud, the same as in Broadbent's case. But this was not all; when the stethoscope was passed, little by little, from the long, rolling mitral bruit towards the ensiform, it was noted that there was a space in which the mitral murmur became lost, while a trifle nearer the sternum another area was reached in which another and shorter presystolic murmur became audible. This fact showed plainly that there were two areas of maximum intensity for these two presystolic murmurs, which fact convinced me that I had to do with two entirely separate and distinct bruits.

It may be objected that this murmur in the epigastrium was the pulmonary regurgitant murmur transmitted to that point. But in reply to this possible objection I need only point out that although both murmurs are diastolic, that of pulmonary regurgitation falls in the early part of diastole immediately after the second sound, while a presystolic one occurs just before the first sound at the end of diastole.

Theoretically and practically, therefore, in endeavouring to establish the existence of tricuspid stenosis, one must search for auscultatory signs in the tricuspid area, yet must remember that owing to the enlargement of the right heart the position of the tricuspid orifice becomes changed, so that the tricuspid area is a wide one. Broadbent detected the murmur in the fifth right interspace, close to the sternum.

Finally, in most cases of this lesion more or less regurgitation is permitted, and hence the tricuspid disease may declare itself by a systolic murmur, the presystolic being either absent or so short as to entirely escape recognition in the presence of the regurgitant bruit. In but 12 of the 154 cases was the tricuspid orifice alone the seat of disease, which shows its rarity apart from associated defects. I should fancy that when it exists alone, it ought to be recognised more easily than when combined, and hence obscured by coexisting disease.

Diagnosis.—Owing partly to the indefiniteness of the physical signs and partly to their being obscured by those of associated valvular lesions, the diagnosis of tricuspid stenosis is generally first made on the autopsy table. Some of the ablest clinical observers believe that an intra-vitam diagnosis must always be problematic, and that when made correctly it is a matter of fortuitous circumstance. One should not feel chagrined, therefore, over his failure to recognise the existence of this disease during life. Conversely, should he be so fortunate as to have his antemortem diagnosis corroborated by the necropsy, he should not take pride to himself, but rather congratulate himself upon the fact that in that particular case the lesion had furnished recognisable physical signs.

Prognosis.—Notwithstanding the fact that one of Leudet's patients is reported to have reached the age of sixty-four, the prospect of long life is not good in cases of this disease. Death overtook the majority of his cases between the ages of twenty and thirty. This brevity of life is due probably not so much to the tricuspid obstruction itself as to its association with other valve-lesions so pronounced as to make it a matter of wonder that patients live as long as they do. When compensation begins to fail there is small prospect of its restoration. The immediate prognosis depends upon the severity of symptoms as well as upon the number and degree of associated lesions. Albuminuria, ascites, hydrothorax, etc., indicate the terminal stage of the disease, and yet proper care and management may extend the life of a patient many months after the urgency of symptoms has compelled him to seek medical aid. Most of the cases reported by Herrick were under observation from a few months to a year or longer.

Mode and Causes of Death.—Death occurred suddenly in one of Herrick's cases, but nothing was found at the post-mortem examination to explain it, further than the ordinary changes in hearts with other valvular diseases. As a rule death comes slowly from gradual cardiac exhaustion, or probably as a result of an approaching standstill in the circulation, due to reversal of the normal blood-pressure within the arterial and venous systems. In Hustedt's single case already mentioned the cause of death was put down as "phthisis," which emphasizes the fact that death is likely to be the indirect rather than the direct result of this valvular defect.

CHAPTER XII

PULMONARY REGURGITATION

THIS form of valvular disease is the corollary of aortic insufficiency, but is infinitely more uncommon, and unassociated with other valvular lesions is very rare. Although I have observed a single instance of chronic organic pulmonary incompetence, as determined by the history and clinical signs, I am indebted for the most of what will be said to Barié's paper on the subject.

Morbid Anatomy.—This depends largely upon the cause of the disease. Thus we recognise two chief groups: (1) A functional or relative incompetence in which the pulmonary artery and ring are so dilated that the valves cannot close the orifice, but are themselves not responsible for the regurgitation. (2) The form in which the leak results from structural changes in the valve-segments. Thus far this disease is the counterpart, both pathologically and etiologically, of the other valvular lesions that have been considered, but the second group of pulmonary regurgitant lesions is again divisible into the congenital and the acquired. In this respect it conforms with what we know of right-heart defects and differs from valvular diseases of the left heart, since congenital affections of the mitral and aortic valves are exceedingly rare.

In relative insufficiency of the pulmonary valve the artery and ostium are found stretched, and the former may show the changes of atheroma, while the leaflets are elongated and broadened in consequence of the strain to which they have been subjected, yet in other particulars are quite likely to be free from disease. This was the case in the patient with mitral and tricuspid stenosis whose history was narrated in the preceding chapter.

In those instances in which the regurgitation is the result of endocarditis, the changes are the same as in other valvular defects of the same origin, and hence do not need to be repeated *in ex-*

tenso. I may only add that the valve-segments have in several cases been found torn into shreds in consequence of inflammatory softening. Not infrequently, according to Barié, the changes are such as to have led to more or less obstruction, as shown in 23 out of 43 cases of pulmonary regurgitation.

Congenital regurgitation at this orifice is certainly rare, and yet Barié is also authority for the statement that it was discovered in 10 out of 34 cases. In this form regurgitation is permitted in consequence of defects in the formation of the valve. In a three and a half months' infant, cited by Barié, there was a rudimentary cusp which allowed a stream of water poured into the artery to leak through into the right ventricle. In Bouillaud's case the orifice was covered by a membranous partition having a circular opening at its centre 6 millimetres in diameter. A mere hint of an attempt at the subdivision of this membrane into segments was shown by the presence of three folds upon its convex aspect.

The secondary effects upon the heart are important. In the congenital cases there is usually discovered a defective closure of the interventricular sæptum or patency of the ductus arteriosus, the same as in congenital pulmonary obstruction. In acquired cases, whether relative or structural, the right ventricle is found dilated, or both hypertrophied and dilated, the same as with the left ventricle in cases of aortic regurgitation. Moreover, in consequence of the easy stretching of the right auriculo-ventricular ring, this orifice is dilated, and the tricuspid valve is also incompetent.

In cases in which pulmonary insufficiency has been the only valvular defect the right heart alone is enlarged, the left chambers being small and looking like mere appendages in comparison with the right. This appearance is not usual, however, because of the coexistence of other valvular disease at the mitral or aortic ostia that have led to secondary enlargement of the left half of the heart. Finally, the myocardium evinces the degenerative effects of long-standing strain and stasis, which have been already described in preceding pages.

Etiology.—In the form of pulmonary regurgitation most frequently recognised—namely, the secondary or relative—the immediate causative element is abnormally high and prolonged blood-pressure in the pulmonary artery. This in turn is due to disease of the left heart or of the lungs. It is in extreme and long-standing mitral stenosis, therefore, that secondary incompetence of the pulmonary valve is oftenest encountered.

Obstruction or regurgitation at the aortic orifice may also produce pulmonary incompetence after having set up stretching of the left auriculo-ventricular ring and relative mitral regurgitation. Great increase of blood-pressure in the pulmonic system is so common and necessary a result of most cardiac and many pulmonary diseases that it is probable that the regurgitation now considered takes place far more frequently than it is recognised clinically.

Of those cases in which the valve itself is diseased the most frequent cause is acute endocarditis. This may be of rheumatic origin, but it is more often septic, and therefore a localization of pus or pneumococcus infection. It occurs in the puerperium, therefore, the same as malignant endocarditis in other situations.

The causes of the congenital form of pulmonary regurgitation are intra-uterine endocarditis and developmental defects, whether due to inflammation or not.

Symptoms.—In those cases in which pulmonary regurgitation is secondary to pre-existing mitral or aortic disease, the symptoms of these latter affections generally obscure those referable to the former, if indeed any new ones are developed. The patients already suffer from the effects of pronounced stasis in the lungs and venous system, and when the pulmonary valves yield to the strain and leak, the force of the regurgitant stream is thrown upon the already overburdened and dilated right ventricle. The propelling power of this portion of the heart is thereby lessened, and the congestion everywhere present is augmented—but as this condition has come on gradually, it is put down as only a further manifestation of the inevitable asystolism.

With greater dilatation of the right ventricle the tricuspid ring stretches and tricuspid regurgitation is added. This leak is more easily recognised than is the pulmonic, and accordingly the still more urgent stasis that now comes on is attributed to the tricuspid incompetence, and the pulmonary insufficiency is overlooked. The seriousness of relative pulmonary incompetence consists, therefore, not so much in the addition of any new and characteristic subjective symptoms as in the fact that it intensifies those already existing, while rendering the prospect of the patient's betterment practically nil.

Objective symptoms—that is, clinical signs of this complication—are present, theoretically at least, and when recognisable consist in a weakening or impurity of the pulmonic second sound, or in a soft diastolic murmur, heard in the second and third left intercostal spaces.

The form which chiefly interests us at this time is the primary or organic, which, either as a congenital or acquired lesion, exists independently of any other valvular or lung disease. Does this produce distinctive subjective symptoms? To this query I think one must reply that not only does it not display distinctive symptomatology, but it sometimes pursues a latent course for many years. In most of the cases cited by Barié the patients displayed dyspnœa and other ordinary tokens of cardiac disease, as cyanosis and venous congestion, that every now and then went on to the production of anasarca, albuminuria, etc., but which were not in any way peculiar.

That the disease may be latent for many years is proved not only by Bouillaud's patient who, in spite of her congenital pulmonary defect, attained the age of twenty-four, but also by a case that came to my notice nearly ten years ago. Most unfortunately an autopsy could not be secured, and hence a post-mortem confirmation of my diagnosis was not had. Yet if physical signs count for anything, then this case, as will be seen by the recital, was one of pulmonary regurgitation unassociated with other valve defects.

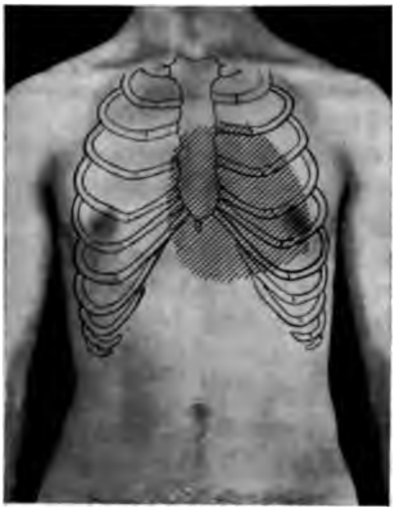


FIG. 75.—AREA OF DEEP-SEATED CARDIAC DULNESS IN CASE OF PULMONARY REGURGITATION (p. 368).

The patient was a married woman of fifty-eight years of age who had given birth to eight children without, she said, any greater difficulty than is experienced by most healthy women.

She was short and slight, and although she stated she had known of her heart-disease since her eighth year, it had never occasioned her any particular discomfort. At the time of her consulting me she had been weak, nervous, and annoyed by palpitations for several weeks, but had objected to seeking medical aid because of prejudice and the discovery, years before, that the ordinary heart medicines did not agree with her.

She showed moderate cyanosis and distention of the external jugular veins, but no œdema, and she did not complain of shortness of breath. The radial pulses were equal, moderately accelerated, irregular in force, and occasionally intermittent. But their particularly noticeable feature was their smallness and feebleness. There was evident, but not great, enlargement of the liver. Thus far there was nothing in the examination of the patient to impress me as unusual.

When, however, exploration of the heart was begun, I was at once struck by the forcible and extensive cardiac impulse, which reached from the left nipple into the epigastrium, quite across the median line to the right costal cartilages, and as far downward as to the level of the eighth. This was not at once recognised as the impulse of the enormously hypertrophied right ventricle, but by carefully studying the apex-beat, and by determining the area of deep-seated cardiac dulness (Fig. 75), I became convinced that it was the right and not the left ventricle which was enlarged.

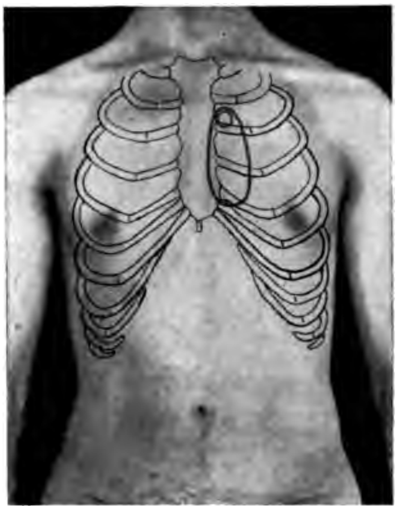


Fig. 76. – Area of Maximum Intensity (small circle) and of Propagation of Murmur in Case of Pulmonary Regurgitation (p. 368).

Then upon resorting to auscultation I at once distinguished a diastolic murmur, which was located at the left of the sternum in the third interspace, was transmitted downward, and possessed the peculiar quality of the aortic regurgitant bruit (Fig. 76). This was quite naturally taken to be aortic, until pondering on the size of the right ventricle and the smallness of the pulse without any suggestion of a collapsing character or of other vascular signs of aortic incompetence, the conviction was at length forced upon me that I had to do with pulmonary regurgitation pure and simple.

I may add that both sounds at the apex were clear, while both second sounds at the base were feeble, and both heart-tones were audible in the cervical arteries. If secondary tricuspid insufficiency existed, it was not recognised. I do not believe it was present, but that the enormous hypertrophy of the right ventricular wall prevented such a degree of dilatation as would have been necessary to set up tricuspid regurgitation.

Not being able to relieve this patient's sense of weakness, nervousness, and inability to take sufficient food, and above all to quiet the violent action of the heart, all of which constituted her symptoms, I was not called in often, and after a few weeks was notified of her death, as nearly as could be determined, from exhaustion.

Here was a patient who to her certain knowledge had been the subject of some form of heart-disease for fifty years, a fact in itself highly interesting and unusual. Moreover, it had not incapacitated her for attending to all the duties of a housekeeper and mother of eight children. And lastly, when the physical signs were carefully determined, they were found to indicate regurgitation into the right and not the left ventricle, consequently pulmonary regurgitation. As no definite and reliable history of diseases in childhood and infancy could be obtained, I was unable to decide whether hers was a congenital or an acquired lesion. It seems to me that there is much matter for reflection in the history of this case and in the long delay of symptoms, which were those of increasing venous stasis, without, however, any complaint of dyspnœa. It would seem to indicate that if this disease is unattended by stenosis, there are no symptoms so long as compensation is maintained, and that this is capable of being preserved for many years.

Physical Signs.—The diagnosis of pulmonary regurgitation concerns both the secondary and the primary form. The former, it will be recollected, is a relative insufficiency depending upon some antecedent pulmonary or cardiac disease, and therefore its physical signs are likely to be obscured by those of the associated affection. The possibility of its occurrence in the late stages of mitral or aortic disease should always be borne in mind, and if in such a case a diastolic murmur develops in the pulmonic area, the diagnosis of secondary pulmonary incompetence may be assumed. Particulars regarding this murmur will be considered under the head of auscultation, to which, therefore, the reader is referred.

Inspection.—This discloses nothing characteristic in the secondary form, the evidences of circulatory embarrassment being due to the associated affections. Even in the primary form the existence of visible signs of heart-disease is likely to depend upon its severity. So long as compensation is preserved, inspection of the præcordia detects nothing more than a forcible and perhaps extended cardiac impulse, which, to judge from the case I have briefly reported, is particularly pronounced in the epigastrium. So soon, however, as the right ventricle begins to fail ocular signs of venous stasis appear, of the same character as in other cardiac affections.

Palpation.—This is of considerable service in the detection of the right ventricle hypertrophy and in the study of the pulse. Pulsation in the epigastrium is forcible and imparts to the palpating hand the impression of a powerfully contracting ventricle. It is in the study of the peripheral arteries that palpation is of greatest value.

Inasmuch as the murmur is so closely like that of aortic regurgitation as to often leave one in doubt concerning its real significance, the pulse must be relied on for differential information. In pulmonary incompetence the aortic system does not experience the sudden distention and equally rapid collapse of aortic insufficiency, and consequently the pulse is not at all like that described by the term collapsing. On the contrary, it is likely to be small and weak, presenting in this respect a striking contrast to what might be looked for in connection with the diastolic murmur at the base of the heart.

The rate and rhythm of the pulse are determined by the state of compensation. If one could place his finger on the pulmonary artery he would discover that this vessel and not the aorta undergoes forcible distention and sudden collapse.

Percussion.—Pulmonary regurgitation affects the size of the right ventricle, and consequently the area of cardiac dulness is increased, chiefly downward, while that at the left is but slightly if at all changed. This means of investigation is therefore of great importance in enabling one to differentiate between pulmonary and aortic incompetence.

In the relative form cardiac dulness is already augmented to the right, and hence percussion is of less value than in the primary variety of this valvular lesion. It may nevertheless be of minor aid in enabling one to determine a degree of enlargement of the right heart out of proportion to what would be expected did mitral or aortic disease exist without pulmonary leakage.

Auscultation.—Contrary to what is usually the case, and to what has been stated in previous chapters concerning the diagnostic value of this means of examination, auscultation is of the greatest assistance in the detection of this particular lesion, not only because of its recognition of a murmur, but also because by it we are able to determine the absence of those vascular phenomena that attend aortic disease of the same nature. Barié directs attention to the importance of carefully studying the pulmonic second tone, since, as he says, the earliest and in some cases the only evidence of relative insufficiency of these valves is to be found in a muffling or impurity of this sound. Consequently, if in a given case of cardiac disease, which ought naturally to intensify the pulmonic second tone, there is heard instead an enfeeblement and trifling impurity of this sound, it should render one suspicious of dilatation of the artery and consequent incompetence of its valve.

If a characteristic murmur results, this is diastolic (Fig. 77), accompanying the second heart-sound or even replacing that usually heard in the second left interspace. In primary lesions this murmur is probably always present, and when stenosis is combined there is also a pulmonic systolic murmur, so that there is a double or to-and-fro bruit, the same as when there is regurgitation at the aortic orifice. The seat of maximum intensity of this diastolic murmur is at the left of the sternum in the second and third left interspaces. Its direction of transmission is downward along the left sternal margin, and its quality is soft. Indeed, it may so closely agree with all the characters of an aortic regurgitant bruit as to make it absolutely essential that one study carefully all the secondary signs before he can arrive at a differential diagnosis. It should be remembered, however, that the pulmonic murmur is not heard at the right of the sternum, as is generally the aortic. Yet this is not alone sufficient for its recognition, since an aortic diastolic murmur is sometimes inaudible at the right and audible at the left of the breastbone. Should a pulmonic systolic murmur be associated, this is not transmitted into the arteries of the neck, but instead the ordinary heart-sounds are there audible, a circumstance which is of diagnostic aid.

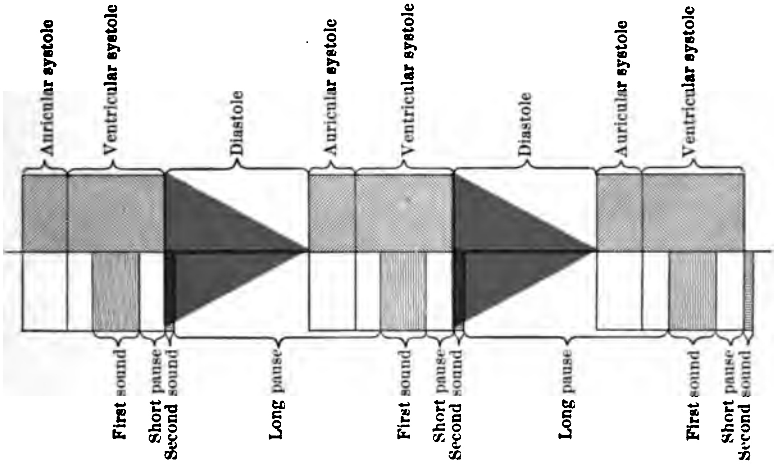


FIG. 77.—RHYTHM OF MURMUR IN TYPICAL CASE OF PULMONARY REGURGITATION.

Diagnosis.—The difficulty which attends the diagnosis of this affection consists not in the recognition of the murmur, but in its interpretation, since it is likely to be mistaken for the bruit of aortic regurgitation. It is indispensable, therefore, to pay attention to the secondary signs, of which the most valuable are those connected with the vascular system. For the reasons stated under palpation, there can be no acoustic phenomena connected with the arterial system in pulmonary insufficiency; and consequently the absence of a systolic snap, and still more of the double souffle in the femorals, would, in conjunction with hypertrophy of the right, not the left ventricle, and with a diastolic murmur at the left cardiac base, enable one to state quite positively that the regurgitation was at the pulmonic, not the aortic ostium. The foregoing remark applies to the relative as well as the primary lesion; for even in cases of combined aortic and mitral incompetence, careful study of the peripheral arteries detects some of the characteristic signs of the former condition. When, on the contrary, mitral disease has led to pulmonary insufficiency, such vascular evidence is wanting.

If the pulmonic valve becomes relatively incompetent in the last stages of aortic stenosis (as I believe occurred in my patient with aortic obstruction whose case was narrated in the chapter on that disease), the recognition of the secondary defect is very difficult. This is so partly because its murmur is faint and likely to be overlooked, but also because if detected it is apt to be attributed to a leak set up for some reason at the aortic orifice. In the case of the lady just alluded to such a diastolic bruit developed some months prior to death, and became attended by distressing and at times violent palpitation at the pit of the stomach, the throbbing being visible. Reflection has since convinced me that this exaggerated action of the right ventricle was an indication of its hypertrophy, and hence corroborative of pulmonary regurgitation. I believe such an observation might be utilized in the future diagnosis of this lesion.

Prognosis.—Very little needs to be said upon this subject. Not only is the disease incurable, but it is not amenable to treatment. In relative pulmonary regurgitation there is already present a disease that affords a grave prognosis, else the pulmonic valve would not give way, and the addition of this complication serves to hasten the downward progress of the patient. It shows that the original affection has reached an extreme stage, and that the right ventricle will not long be able to withstand the strain.

If the pulmonary incompetence is unattended by other cardiac disease, and if compensatory hypertrophy is good, the lesion being discovered accidentally perhaps, the regurgitation may last for years without producing serious circulatory disturbance, as shown by my patient who had the lesion fifty years. This freedom from symptoms is the exception, however, for the wall of the right ventricle is so thin that compensatory hypertrophy is likely to be easily ruptured, and when subjective symptoms once set in they are likely to be progressive. The occurrence of albuminuria and dropsy is to be regarded as of very evil import and to betoken the not very remote termination of the case.

Mode and Causes of Death.—Barié speaks of the occurrence of pulmonary embolisms as a not remote contingency and as contributing to the patient's death. The fatal termination is most likely to supervene slowly, in consequence of cardiac or general exhaustion, rather than suddenly, and yet this latter event is not impossible. Congenital cases, particularly if combined with stenosis, may lead to pulmonary phthisis through anæmia of the lungs.

CHAPTER XIII

PULMONARY STENOSIS

This is an obstructive lesion which in its effect on the right ventricle is analogous to that of aortic stenosis on the left. It differs from the latter, however, in its origin and anatomical characters. It is divided into two great classes, the congenital and the acquired, the former constituting by far the greater number. Even when congenital, this lesion is one of the rare forms of heart-disease. Instances of the acquired affection are so extremely infrequent that since Constantin Paul's elaborate monograph in 1871 only 8 cases have, so far as I know, been reported. For my knowledge of these cases I am indebted to a thesis by Koehler, of Halle, in 1894. I have not been able to ascertain how many of the cases described by Paul belong to each variety. The 3 cases reported in 1895 by Boviard, Holt, and Forlanini, and the 3 in 1896 by Adams, Arnozan, and Siredy, all appear to have been congenital, which still further emphasizes the rarity of the acquired form. The first case on record of this form was described in the Atlas of Pathological Anatomy by Cruveilhier.

Morbid Anatomy.—This can be best described by the repetition of a report of the 8 cases above alluded to, which was published by me in the Journal of Medicine in January, 1897.

In 1873 the late Christian Fenger published a case in a male of nineteen years in which the disease was traceable to an attack of articular rheumatism at the age of eleven. The autopsy disclosed numerous vegetations attached to the pulmonary valve and along the wall of the artery as far as its bifurcation and into its main branches, particularly the left. This condition, although the vessel was dilated, had led to very great obstruction, with consecutive hypertrophy of the right ventricle. The septa were intact, the pulmonary artery and its main right branch dilated. Fenger, from the history and post-mortem discovery of recent endocarditis, concluded that there could be no doubt of the postnatal origin of this case.

Moritz Mayer next reported a case in 1874 in a girl of sixteen, who at the age of eleven had suffered from some pulmonary disease and from endocarditis. The necropsy revealed cauliflower vegetations attached to the wall of the conus arteriosus and to the pulmonary valves, with secondary hypertrophy and dilatation of the right ventricle. The ductus arteriosus was closed, but the interventricular sæptum contained an opening large enough to admit the tip of the first finger, and Mayer explained this defect as having originated after birth in consequence of the previous endocarditis.

Rinsenna's case in 1883 was in a patient aged thirty-four, who had also had acute rheumatism. On post-mortem examination the pulmonary valves were found greatly thickened, and to have thus caused slight obstruction, but without enlargement, of the right ventricle.

In 1884 Krannhals reported 2 cases, of which one was in a widow of forty-three suffering from leucæmia, and the pulmonary stenosis was attributed to this affection. Neither necrosis of the valves nor micro-organisms were found to explain the stenosis. In his second case, that of a housemaid of nineteen, there was a history of good health up to the fourth year, when she had an attack of rheumatism, and her health had been impaired since that time. The fœtal passages were found closed, there were no congenital defects, but the pulmonary artery was dilated.

Rendu in 1884 described a case in a girl of nineteen, who had had the disease for fifteen years, and after death the pulmonary valves were found fused into an inflexible diaphragm having an opening of about $\frac{1}{10}$ of an inch in diameter. The immediate cause of death was a rapidly progressing nephritis.

Stybr's case in 1890 was that of a woman of twenty-four in whom the stenosis was found due to an inflammatory blending of the pulmonary segments into a tendon-like cone that projected into the lumen of the artery and contained at its apex an opening 2 millimetres in width. Two oblique lines that passed down the sides of the cone showed where the cusps had become united. The right ventricle was enormously hypertrophied. As the aortic cusps were slightly sclerosed, Koehler thinks the inflammatory process must have dated from intra-uterine life.

Finally, Koehler described the case of a housemaid of twenty-one who was admitted to the hospital in July, 1893, suffering from pneumonia, and who gave a history of acute rheumatism the May previous. Since that time her health had been poor, and she had suffered from dyspnœa. The autopsy disclosed polypoid vegetations springing from the wall of the pulmonary artery in such a manner as to prevent the complete opening of the valve during ventricular systole. The valve-segments as well as the endocardium of the right ventricle were healthy, but similar vegetations were found in the aorta so disposed as to prevent the adequate opening of these valves, and to thus cause a stenosis of this orifice, the same as on the right side. Both ventricles, particularly the right, were dilated. The sæptum was intact and the foramen ovale was closed.

In the foregoing cases, with exception of Rendu's and Stybr's, there were evidences of recent endocarditis, absence of congenital abnormalities, and dilatation, or at least no perceptible narrowing, of the pulmonary artery. Acquired cases present striking differences from most of the congenital in their pathological appearances, but they both have the common feature of hypertrophy and dilatation of the right ventricle.

In the congenital form the obstruction is due most usually to a fusing together of the valve-segments, which then form either a diaphragm stretching across the ostium or a cone-like projection into the artery with a small opening at its apex (Fig. 78). The foramen ovale is usually open, and the interventricular sæptum is sometimes incomplete. The pulmonary artery is always narrowed, and there may be atresia of this vessel. The ductus arteriosus is generally open, yet is in some cases found closed. Very rarely the stenosis is caused by constriction of the right conus arteriosus, in which event this may appear like a third ventricle, and both the interventricular and interauricular septa are defective, the pulmonary artery is narrowed or even occluded, and the ductus Botalli remains pervious.

In other cases there are various errors of development, as transposition of the aorta and pulmonary artery, or their origin from one common trunk; a blending of the two ventricles into one common cavity with but one instead of two auricles; or one ventricle and two auricles, or but one auricle and two ventricles.

It is not always easy to determine post mortem whether a given case belongs to the congenital or the acquired category. In well-marked specimens, like Fenger's, or in such as show striking abnormalities of development the decision may be easy; but in cases presenting some of the characteristics of both forms the exact nature must be left in doubt.

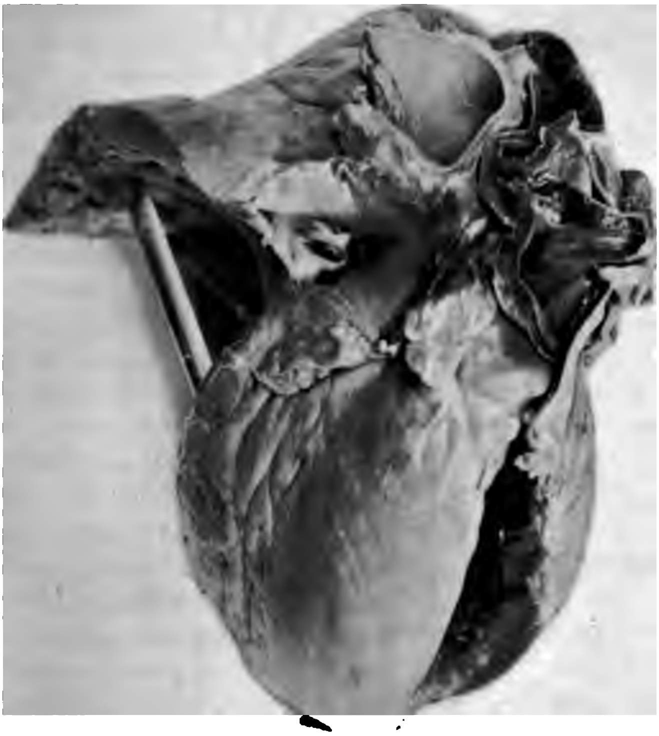


Fig. 78.—Heart of a Boy, showing Congenital Stenosis of the Pulmonary Orifice. Specimen in collection of Dr. Gustav Futterer.

Aside from the evidences of recent endocarditis it is the condition of the pulmonary artery upon which pathologists rely for the determination of the intra- or extra-uterine development of the lesion. Dilatation of this vessel makes strongly for the acquired form, while narrowing of the artery points to the fœtal origin of the disease.

Etiology.—The congenital form can be dismissed with the statement that it results either from intra-uterine endocarditis or myocarditis, or from defective development.

Acquired cases also originate in endocarditis either of rheumatic causation or in the course of other acute infectious processes, as shown in the histories of the 8 cases above narrated. Its rarity is due to the fact that after birth the right heart is seldom the seat of acute inflammation.

In a ninth case that has come to my notice, that of A. Kasem-Bek in 1899, the pulmonary stenosis was caused by a gumma on the ostium.

Symptoms.—These depend largely upon the congenital or acquired nature of each case. Moreover, in the latter the clinical history is also influenced by the presence or absence of acute endocarditis. If the cases are not stumbled upon accidentally in the course of examination or treatment for some other wholly different disease, the patients are likely to be seen when the affection has led to pronounced disturbance of the general health. In such there are dyspnœa and other ordinary evidences of cardiac asthenia, or there is a complaint of vague general distress and ill health. In a word, there are no symptoms peculiar to pulmonary stenosis as contrasted with other valvular lesions.

In the congenital form patients are apt to be weakly, undersized, sometimes mentally deficient, and to manifest striking cyanosis. This is not always present, however. In the chapter on Congenital Cardiac Affections will also be considered certain changes in the blood that accompany marked cyanosis or the *Morbus Ceruleus* of older writers.

Sufferers from pulmonary stenosis *are very apt to die from tuberculosis of the lungs*, as is shown in the only instance of this cardiac disease I have observed, and which was published in my paper previously mentioned. A plumber's helper, aged twenty-three, was first seen by me at my clinic at Cook County Hospital, having been sent from Ward 4 as a case of pulmonary tuberculosis. Family history was meagre. His father had died of some wasting disease with cough; his mother of cancer; two sisters living and healthy. Patient declared he was healthy in infancy and childhood, and had never suffered from dyspnœa on exertion prior to his present illness, and had not exhibited cyanosis. In fact he was healthy until his present illness began three months before his admission to the hospital.

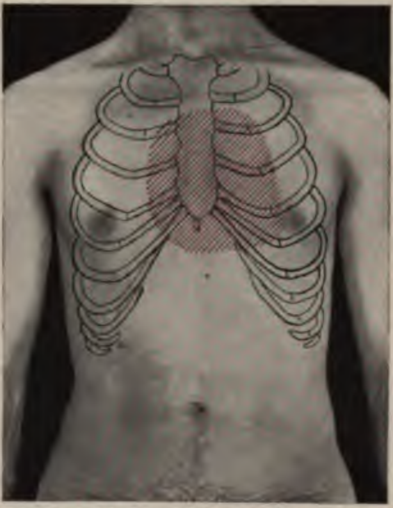


FIG. 79.—RELATIVE CARDIAC DULNESS IN CASE OF PULMONARY STENOSIS (p. 380).

Without entering too much into detail, it will suffice to state that he presented the usual symptoms of consumption, emaciation, cough, profuse muco-purulent expectoration, febrile temperature, and a rapid, feeble pulse, the functions of the digestive organs remaining good. There was no cyanosis. The right apex was retracted, and expanded poorly upon inspiration. Both apices showed dulness, bronchial breathing, and moist râles.

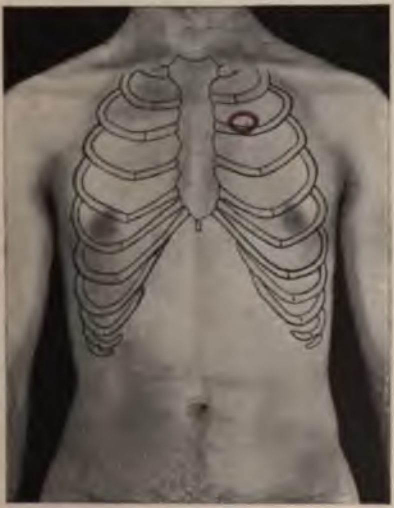


FIG. 80.—LOCATION OF THRILL AND SYSTOLIC MURMUR IN CASE OF PULMONARY STENOSIS (p. 380).

The præcordium bulged from the third rib to the epigastrium and from left to right nipple. There was a short, weak systolic thrill in second left interspace, 1 inch from sternum. Absolute dulness was increased from the level of the second costal cartilage to the lower border of the fifth rib, and from 1½ inch to right of sternum to ¼ inch inside of left mamillary line (Fig. 79).

The heart's rhythm was regular and accelerated, the sounds being very feeble, the first muffled and dull, while the second, in the third left interspace, was short, high-pitched, and so feeble as to be rudimentary. A harsh systolic murmur was audible, having its maximum intensity in the second left interspace, 1 inch from sternum, and corresponding in position to the soft systolic thrill previously mentioned (Fig. 80). It was transmitted with special clearness upward and outward towards the left shoulder and around the left side to the back, but could be distinguished feebly even in the right half of the thorax. The liver was not appreciably enlarged. Tubercle bacilli were discovered in the sputum.

The diagnosis was made of pulmonary stenosis with secondary hypertrophy and dilatation of the right ventricle; tuberculosis of both lungs and moderate venous and visceral congestion consecutive to the cardiac lesion. This was thought to be congenital, although there was no history of cyanosis in infancy, and no evidence of other congenital cardiac defects. The patient was kept under observation until January 10, 1896, when he was found dead in his bed. Symptoms of general asthenia increased in severity, and diarrhœa set in a day or two before death.

Necropsy was made by Dr. F. Tice twenty-four hours after death. The lung-findings, briefly stated, were those of pulmonary tuberculosis. The pericardium contained from one and a half to two ounces of fluid. Aorta was not enlarged; the pulmonary artery was larger than the aorta, dilatation extending into the two branches, the left more than the right. The aortic valves were found competent, but the pulmonary valves leaked slowly to the hydrostatic test.

Looking into the pulmonary artery from above, it appeared as if a nipple with a small opening at its apex projected into the vessel, and at one side near its base was a second small opening, which was closed in below by a thinner membrane (Fig. 81). The right ventricle was hypertrophied and dilated, and the right auricle was also enlarged. One cusp of the tricuspid valve showed a slight thickening along its base. The mitral valves were negative except a slight thickening; aortic valves were thickened along base and margins, while small atheromatous plaques were found in the beginning of the aorta.

The left ventricle appeared slightly dilated. The interventricular sæptum was complete, but in the interauricular sæptum there was a valve-like passage, which would not quite admit two matches, was perhaps 3 millimetres in diameter, and corresponded in situation and shape to the foramen ovale (Fig. 82). Thus it was seen that the intra-vitam diagnosis was confirmed in its main features. A more careful inspection of the heart, made a year later after having been preserved in a formalin solution, showed that the cone which projected into the pulmonary artery, and represented the semilunar valves, was made up of a uniform membrane, somewhat thicker than normal valves, and showed no lines or ridges that indicated the points of fusion of the cusps. The opening at the apex was oval, measuring 15 millimetres by 8 millimetres at its broadest point. The edges of the cone were thickened, and the second minute opening at its side, near its base, was

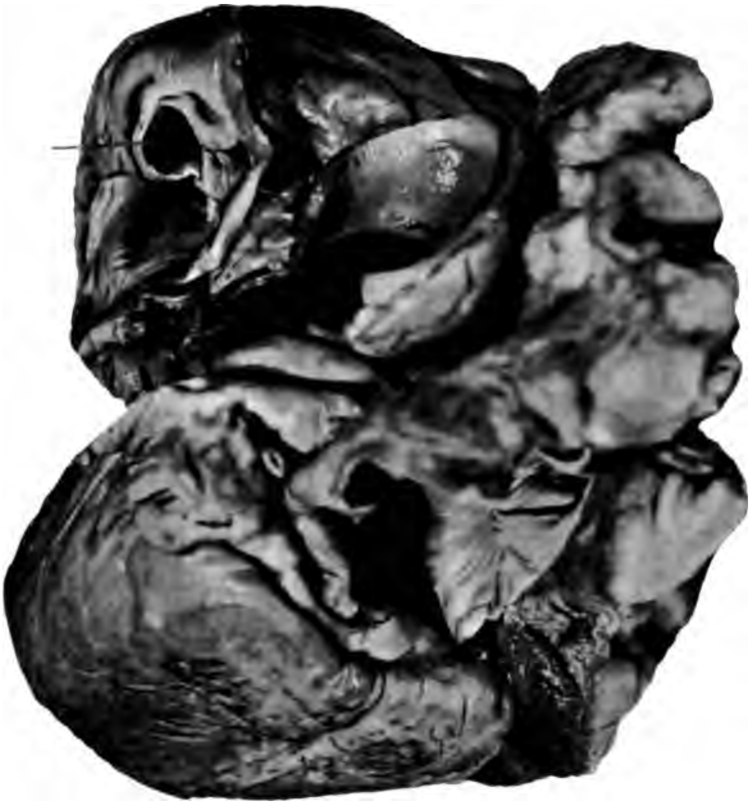


Fig. 81.—Heart from Case of Pulmonary Stenosis (p. 380).
Line shows narrowed pulmonary orifice.

found to be a saccular dilatation projecting into the lumen of the cone. The diameter of the pulmonary artery was 22 millimetres, of the aorta 18, and of the foramen ovale 3 millimetres.

From the foregoing description it is apparent with what uncertainty one can classify this case as congenital or acquired. There were no indications of endocarditis, as it ordinarily appears after birth. The pulmonary artery was not contracted, but rather dilated, the interventricular septum was complete, and the foramen ovale was not more patent than it is in a considerable proportion of hearts without a suspicion of congenital disease. Nevertheless, the appearance of the cone, which replaced the semilunar

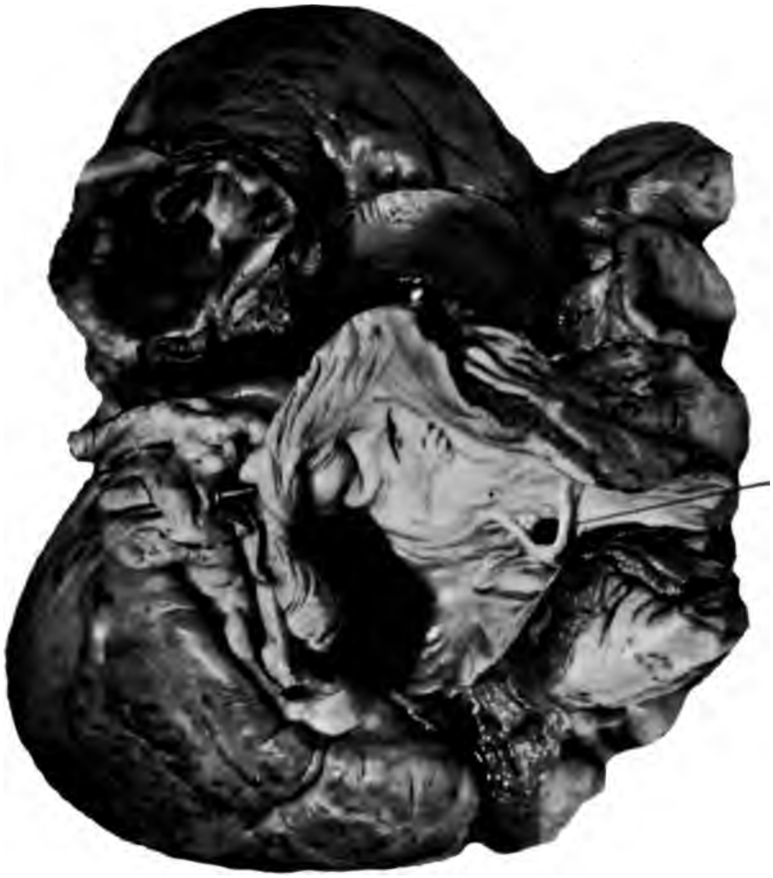


Fig. 82.—Same Heart as Fig. 81.

Left auricle is laid open, and line indicates patent foramen ovale.

valves, rendered it probable that this case was of congenital origin, and that intra-uterine endocarditis caused a fusion of the segments at a period subsequent to the closure of the septa, and that the stenosis was not sufficient to prevent the closure of the foramen ovale after birth. Moreover, the volume of blood driven into the pulmonary artery could not have been very small, and must have been divided into fluid veins, which threw the stream against the arterial coats in such a way as to maintain adequate blood-pressure within the vessel. It is interesting to reflect that, although the lesion, according to the patient's history, gave rise to no subjective symptoms, it should yet ultimately have led to pulmonary tuberculosis, the usual sequence in such cases.

Regarding physical signs in this case, it is also of interest to note the wide propagation of the murmur. This was more marked in the left lung, and as the left branch of the pulmonary artery was found to be rather larger than the right, there was probably direct connection between the size of the artery and the transmission of the murmur; for had the vessel been narrowed, the audible vibrations could not have been transmitted to any great distance.

If a conclusion from a single case is justifiable, it is likely that the symptoms directly referable to pulmonary stenosis depend upon its degree and upon the association of other developmental defects even more than upon the obstruction itself.

Physical Signs.—*Inspection.*—Cyanosis is not always present in congenital cases, and when present is not uniform throughout the body. It is this bluish tint which led old writers to designate the underlying abnormality by the generic term of Morbus Ceruleus. Cyanosis is most apparent on the cheeks, ears, fingers, elbows, and knees, and is intensified by coughing or physical exertion. As shown by my case, it is not so likely to be present when the stenosis has not led to or is not associated with defects, as, e. g., patency of the foramen ovale or of the interventricular sæptum.

In the acquired form patients are more apt to show pallor of the countenance, and there may be turgescence of the superficial veins as a direct result of interference with the outflow of blood from the right heart. Inspection of the præcordium detects bulging over the situation of the right ventricle—i. e., at the lower end of the sternum and the parts immediately adjacent. Such a bulging is most marked in cases in which the valvular lesion is either congenital or has existed from very tender years. It is due to hypertrophy of the right ventricle, and hence there is associated pulsation in the epigastrium and over the prominent area.

Palpation.—This corroborates some of the information derived by inspection, and enables one still better to appreciate the extent and force of the cardiac impulse imparted by the hypertrophied right ventricle. In addition, there is usually felt a systolic purring vibration or thrill in the pulmonic area—i. e., in the second left intercostal space, close to the sternum. This fremissement may be exceedingly delicate, as in the case observed by me, or it may be distinct and harsh.

The pulse presents no distinctive characters aside from smallness, feebleness, and increased frequency.

Percussion.—By this means is revealed marked increase of both absolute and relative cardiac dulness, the increase being downward and to the right, over the situation of the right ventricle and corresponding auricle (Fig. 79). In my case this alteration of cardiac dulness was very pronounced, and aided materially in the diagnosis of the lesion by assuring me of the existence of right ventricle hypertrophy.

Auscultation.—The conditions here are favourable to the generation of a bruit, and as it is produced during the passage of the blood from the right ventricle into the pulmonary artery, the murmur is systolic (Fig. 83). Its seat of maximum intensity is over the base of the heart, at the left of the sternum, in the second and third intercostal spaces (Fig. 80). At first this bruit may be thought to be aortic in origin, but if its direction of propagation is studied this will be found to be upward and to the left towards the left clavicle rather than to the right and upward, as is the case with the murmur of aortic stenosis. Another point of difference is that the pulmonic systolic murmur is not heard in the cervical arteries, where, on the contrary, the two cardiac tones are usually distinct. If intense, the murmur may be heard throughout the præcordium, though in all instances its area of maximum intensity corresponds with the location of the systolic thrill. In my case the murmur was transmitted widely, but was more distinct in the left than in the right half of the thorax. In auscultating towards the apex and over the body of the right ventricle the murmur grows less intense, while the two cardiac sounds become more distinct. The murmur may be rough and loud, or soft and faint. The pulmonic second sound is diminished in intensity, or may be absent altogether, while the two aortic sounds are distinct.

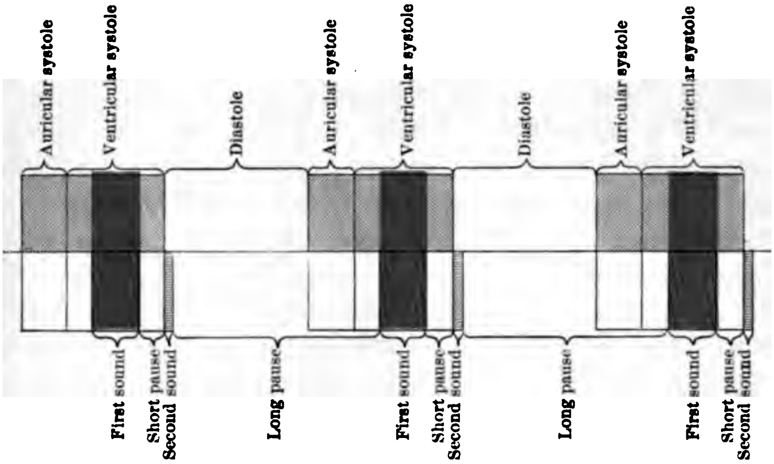


FIG. 83.—RHYTHM OF TYPICAL PULMONARY STENOTIC MURMUR.

Diagnosis.—The difficulty of diagnosis in this affection lies in its differentiation from aortic stenosis or possibly in deciding whether the bruit may not be accidental, since it is situated where such an accidental murmur is so often heard. If, however, proper attention is paid to the secondary physical signs as described above, in particular to the evidence of hypertrophy of the right, not of the left ventricle, one can scarcely fail to interpret the murmur correctly. If after such careful study of all the physical signs doubt is still entertained, the sphygmograph will be found of service in enabling one to differentiate between this lesion and aortic stenosis, for it goes without saying that pulmonary obstruction can in nowise modify the characters of the radial pulse. Finally, the discovery of pulmonary tuberculosis and of cyanosis in cases in which the two halves of the heart communicate, furnishes a certain degree of evidence in favour of the existence of pulmonary obstruction.

Prognosis.—Patients with pulmonary stenosis rarely live beyond the third decade. Even when the disease does not directly destroy life, it does so indirectly by predisposing to pulmonary tuberculosis. This has been so frequently observed that no doubt can be entertained concerning the intimate connection existing between these two diseases. When phthisis has once supervened the prognosis becomes that of the secondary affection, and since the stenosis is irremovable there can be no hope of the arrest of the tuberculosis. The influence of this cardiac defect upon the production of consumption is through the anæmia of the lungs which the narrowing of the ostium occasions.

Mode and Causes of Death.—In Hustedt's 6 cases of pulmonary stenosis death was caused in 1 case by heart weakness, in another by miliary tuberculosis, and in the 4 others by phthisis.

Summary of Physical Signs of Valve Lesions of the Right Heart

	Apex-beat.	Heart dulness.	Murmurs.	Secondary signs.	Pulse.
Tricuspid regurgitation.	Usually feeble and displaced or formed by impulse of hypertrophied right ventricle	Increased to right and downward. Sometimes also to left.	Systolic in tricuspid area. Those also of associated or primary lesions.	Positive venous pulse in jugulars and liver.	That of the primary affection usually small and weak.
Tricuspid stenosis.....	Of left ventricle variable; thumping in tricuspid area (epigastrium) with or without presystolic thrill.	Increased greatly to right of sternum, perhaps also downward. And often also to the left.	Not constant. A presystolic, often with systolic in tricuspid area, frequently obscured by other murmurs.	Those of great venous engorgement. May be thumping, shock, and thumping first sound in tricuspid area.	Small and weak, not characteristic.
Pulmonary regurgitation.	Of left ventricle weak. Strong heaving pulsation in epigastrium.	Increased to right and downward. Also to left in secondary cases.	Soft diastolic in pulmonary area with or replacing second sound. Transmitted downward.	Those of hypertrophy of right ventricle.	Small, weak, not collapsing.
Pulmonary stenosis....	Of left ventricle weak. Strong pulsation of right ventricle in epigastrium.	Increased to the right and chiefly downward.	Systolic in pulmonary area, transmitted upward to left.	Systolic thrill at site of murmur. Feeble or absent pulmonic second sound; possibly cyanosis or signs of secondary pulmonary tuberculosis.	Small and weak, not characteristic.

CHAPTER XIV

COMBINED VALVULAR LESIONS

CHRONIC valvular defects have been dealt with singly, since by so doing their distinctive individual features could be brought out more clearly and without danger of confusion. It would be a mistake, however, to consider these forms of heart-disease as always, or indeed as generally, occurring alone. As a matter of fact certain of them are usually combined, while it is possible for any two or three, or even for all of them to be united. The most common association is that of both stenosis and regurgitation at the same orifice. Thus it is comparatively rare to find aortic obstruction without also some insufficiency, or the reverse, while in the same manner the two mitral lesions are usually combined in varying proportions. Indeed, a moment's reflection will convince one that the structural alterations set up by endocarditis are very prone to result in both incompetence of the valves and narrowing of the ostium, the clinical features of each case being determined by the predominant lesion.

This is not all; lesions at one orifice may be complicated by a defect situated at another. To be explicit, mitral stenosis may be combined with either aortic stenosis or regurgitation, or both, and the same way with mitral insufficiency, or a double mitral disease may be associated with either or both of the aortic defects. Let us now consider these various combinations in detail.

COMBINED MITRAL STENOSIS AND REGURGITATION

As already stated, the endocarditic changes that lead to mitral disease are very apt to cause both constriction and insufficiency. Extreme narrowing is more likely to exist alone than is free regurgitation, and yet even when there is a buttonhole mitral, it is possible for an insignificant leak to also occur, although the insufficiency may not be declared by a systolic apex-murmur. On the other hand, mitral segments that are too stiff to close are quite likely to depend in front of the opening in such a manner as to oppose some barrier to the free ingress of the blood from the auricle. In children the mitral curtains are not infrequently so shrivelled as to form a mere fringe about the ring, and when such is the case stenosis is absent. In adults, particularly when the incompetence is the result of atheroma, pure and unmixed regurgitation is the exception.

The effects on the heart are essentially those of either form of mitral disease when existing alone, and yet the left ventricle and left auricle manifest certain modifications depending upon the association of stenosis with incompetence. The ventricle is neither so dilated as in unmixed regurgitation, nor so atrophic as when there is extreme obstruction. Similarly, the left auricle is neither so hypertrophied as in predominating stenosis, nor so dilated as in free regurgitation. When conjoined, the two lesions, therefore, exert a somewhat restraining influence upon each other as regards the secondary effects on the cardiac cavities directly affected. The changes in the right heart are those incident to retarded pulmonary circulation, and their extent depends upon which of the two lesions predominates.

Symptoms.—The symptoms depend upon the degree of compensation, and this on whether the stenosis or the regurgitation is the greater. They have been described in considering the respective mitral defects, and do not need to be recapitulated.

Diagnosis.—The diagnosis is as a rule not difficult, for the reason that the signs of the two diseases are combined with varying distinctness in different cases. The apex-beat is not so displaced nor so forcible as in uncomplicated regurgitation, nor, on the other hand, is it so distinctly thumping as in pure stenosis, but presents the characters of both affections. There is usually a presystolic thrill at the apex, but it is less long and less intense than in stenosis alone, being commonly only a short vibration, which seems to be merely a prolongation of the apex-shock. Cardiac dulness is increased transversely, but chiefly to the right, and the pulmonic second sound is accentuated.

Auscultation detects a combination of both a presystolic and a systolic murmur, the latter being well marked as a rule, and the former long and relatively pronounced, or short and difficult of recognition, according to the degree of narrowing. I have sometimes found in these cases that the systolic bruit is the predominant one in the erect position, while in the dorsal decubitus the presystolic murmur comes out more distinctly. This is the reverse of what has been stated as the rule regarding the influence of position upon the two mitral murmurs when uncombined. I have also observed that often, when only the presystolic bruit is audible directly at the apex, the systolic murmur can be detected further to the left and on the back.

Prognosis.—The prognosis is, other things being equal, rather more favourable when these two conditions are united than when either exists alone, and I believe for the reason that they tend to check each other.

MITRAL STENOSIS AND AORTIC STENOSIS

This is an exceedingly serious combination, since at both of the left ostia there is a mechanical impediment to the passage of blood from the pulmonary into the aortic system. The left ventricle receives and discharges an abnormally small volume of blood, depending on the degree of constriction, and hence is a thick-walled chamber of limited capacity, while in marked contrast are the greatly hypertrophied and dilated left auricle and right ventricle.

Symptoms.—Symptoms appear early, and are pronounced in consequence of the great stasis within the lungs and body generally. Cyanosis and dyspnœa are present, often in an extreme degree, while engorgement of the abdominal and pelvic organs is shown by all of its attendant phenomena, both subjective and objective.

Diagnosis.—The pulse is small, weak, and slow or accelerated, according to the state of compensation. The apex-beat is weak and preceded by a presystolic thrill, unless indeed it be produced by the impulse of the hypertrophied right ventricle, when it may be diffused and quite strong between the sternum and left nipple.

Epigastric pulsation and a marked increase of absolute and relative cardiac dulness to the right evince the secondary enlargement of the right heart. There are heard a rough, low-pitched presystolic murmur at or within the apex and an accentuated pulmonic second sound indicative of the mitral lesion, and in addiless intense, while the two cardiac sounds become more distinct. The murmur may be rough and loud, or soft and faint. The pulmonic second sound is diminished in intensity, or may be absent altogether, while the two aortic sounds are distinct.

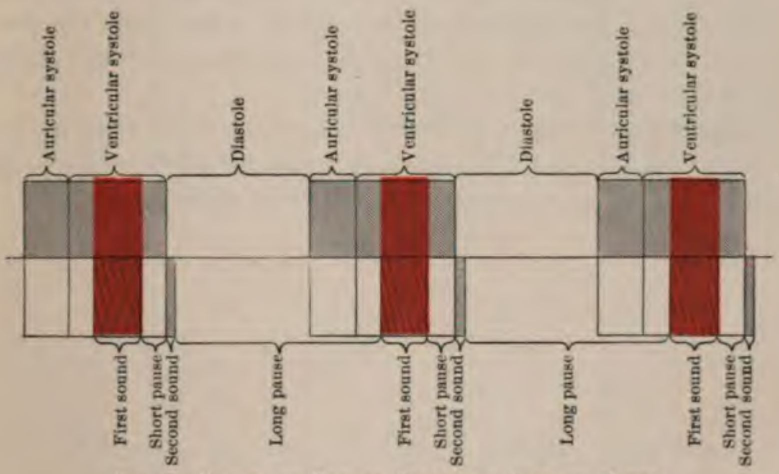


FIG. 83.—RHYTHM OF TYPICAL PULMONARY STENOTIC MURMUR.

Diagnosis.—The difficulty of diagnosis in this affection lies in its differentiation from aortic stenosis or possibly in deciding whether the bruit may not be accidental, since it is situated where such an accidental murmur is so often heard. If, however, proper attention is paid to the secondary physical signs as described above, in particular to the evidence of hypertrophy of the right, not of the left ventricle, one can scarcely fail to interpret the murmur correctly. If after such careful study of all the physical signs doubt is still entertained, the sphygmograph will be found of service in enabling one to differentiate between this lesion and aortic stenosis, for it goes without saying that pulmonary obstruction can in nowise modify the characters of the radial pulse. Finally, the discovery of pulmonary tuberculosis and of cyanosis in cases in which the two halves of the heart communicate, furnishes a certain degree of evidence in favour of the existence of pulmonary obstruction.

Prognosis.—Patients with pulmonary stenosis rarely live beyond the third decade. Even when the disease does not directly destroy life, it does so indirectly by predisposing to pulmonary tuberculosis. This has been so frequently observed that no doubt can be entertained concerning the intimate connection existing between these two diseases. When phthisis has once supervened the prognosis becomes that of the secondary affection, and since the stenosis is irremovable there can be no hope of the arrest of the tuberculosis. The influence of this cardiac defect upon the production of consumption is through the anæmia of the lungs which the narrowing of the ostium occasions.

Mode and Causes of Death.—In Hustedt's 6 cases of pulmonary stenosis death was caused in 1 case by heart weakness, in another by miliary tuberculosis, and in the 4 others by phthisis.

Summary of Physical Signs of Valve Lesions of the Right Heart

	Apex-beat.	Heart dulness.	Murmurs.	Secondary signs.	Pulse.
Tricuspid regurgitation.	Usually feeble and displaced or formed by impulse of hypertrophied right ventricle	Increased to right and downward. Sometimes also to left.	Systolic in tricuspid area. Those also of associated or primary lesions.	Positive venous pulse in jugulars and liver.	That of the primary affection usually small and weak.
Tricuspid stenosis......	Of left ventricle variable; thumping in tricuspid area (epigastrium) with or without presystolic thrill.	Increased greatly to right of sternum, perhaps also downward. And often also to the left.	Not constant. A presystolic, often with systolic in tricuspid area, frequently obscured by other murmurs.	Those of great venous engorgement. May be thumping, shock, and thumping first sound in tricuspid area.	Small and weak, not characteristic.
Pulmonary regurgitation.	Of left ventricle weak. Strong heaving pulsation in epigastrium.	Increased to right and downward. Also to left in secondary cases.	Soft diastolic in pulmonary area with or replacing second sound. Transmitted downward.	Those of hypertrophy of right ventricle.	Small, weak, not collapsing.
Pulmonary stenosis....	Of left ventricle weak. Strong pulsation of right ventricle in epigastrium.	Increased to the right and chiefly downward.	Systolic in pulmonary area, transmitted upward to left.	Systolic thrill at site of murmur. Feeble or absent pulmonic second sound; possibly cyanosis or signs of secondary pulmonary tuberculosis.	Small and weak, not characteristic.

CHAPTER XIV

COMBINED VALVULAR LESIONS

Chronic valvular defects have been dealt with singly, since by so doing their distinctive individual features could be brought out more clearly and without danger of confusion. It would be a mistake, however, to consider these forms of heart-disease as always, or indeed as generally, occurring alone. As a matter of fact certain of them are usually combined, while it is possible for any two or three, or even for all of them to be united. The most common association is that of both stenosis and regurgitation at the same orifice. Thus it is comparatively rare to find aortic obstruction without also some insufficiency, or the reverse, while in the same manner the two mitral lesions are usually combined in varying proportions. Indeed, a moment's reflection will convince one that the structural alterations set up by endocarditis are very prone to result in both incompetence of the valves and narrowing of the ostium, the clinical features of each case being determined by the predominant lesion.

This is not all; lesions at one orifice may be complicated by a defect situated at another. To be explicit, mitral stenosis may be combined with either aortic stenosis or regurgitation, or both, and the same way with mitral insufficiency, or a double mitral disease may be associated with either or both of the aortic defects. Let us now consider these various combinations in detail.

COMBINED MITRAL STENOSIS AND REGURGITATION

As already stated, the endocarditic changes that lead to mitral disease are very apt to cause both constriction and insufficiency. Extreme narrowing is more likely to exist alone than is free regurgitation, and yet even when there is a buttonhole mitral, it is possible for an insignificant leak to also occur, although the insufficiency may not be declared by a systolic apex-murmur. On the other hand, mitral segments that are too stiff to close are quite likely to depend in front of the opening in such a manner as to oppose some barrier to the free ingress of the blood from the auricle. In children the mitral curtains are not infrequently so shrivelled as to form a mere fringe about the ring, and when such is the case stenosis is absent. In adults, particularly when the incompetence is the result of atheroma, pure and unmixed regurgitation is the exception.

The effects on the heart are essentially those of either form of mitral disease when existing alone, and yet the left ventricle and left auricle manifest certain modifications depending upon the association of stenosis with incompetence. The ventricle is neither so dilated as in unmixed regurgitation, nor so atrophic as when there is extreme obstruction. Similarly, the left auricle is neither so hypertrophied as in predominating stenosis, nor so dilated as in free regurgitation. When conjoined, the two lesions, therefore, exert a somewhat restraining influence upon each other as regards the secondary effects on the cardiac cavities directly affected. The changes in the right heart are those incident to retarded pulmonary circulation, and their extent depends upon which of the two lesions predominates.

Symptoms.—The symptoms depend upon the degree of compensation, and this on whether the stenosis or the regurgitation is the greater. They have been described in considering the respective mitral defects, and do not need to be recapitulated.

Diagnosis.—The diagnosis is as a rule not difficult, for the reason that the signs of the two diseases are combined with varying distinctness in different cases. The apex-beat is not so displaced nor so forcible as in uncomplicated regurgitation, nor, on the other hand, is it so distinctly thumping as in pure stenosis, but presents the characters of both affections. There is usually a presystolic thrill at the apex, but it is less long and less intense than in stenosis alone, being commonly only a short vibration, which seems to be merely a prolongation of the apex-shock. Cardiac dulness is increased transversely, but chiefly to the right, and the pulmonic second sound is accentuated.

Auscultation detects a combination of both a presystolic and a systolic murmur, the latter being well marked as a rule, and the former long and relatively pronounced, or short and difficult of

[illegible]

[illegible] [illegible]

MITRAL STENOSIS AND AORTIC STENOSIS

[illegible]

Symptoms [illegible]

Diagnosis [illegible] ventricle, when [illegible] sternum and left [illegible]

[illegible] of absolute and [illegible] right [illegible] the secondary enlargement [illegible] There are heard a rough, low-pitched [illegible] the apex and an accentuated pulmonic second sound indicative of the mitral lesion, and in addition, a harsh systolic bruit in the aortic area with a feeble second tone, showing obstruction at this orifice.

Prognosis.—The prognosis is of necessity very unfavourable, since compensation cannot long be preserved, and when broken can be restored, if at all, only with great difficulty.

MITRAL STENOSIS AND AORTIC REGURGITATION

This is also a serious combination, yet the degree of its gravity is determined by the extent and predominance of the lesions.

The secondary effects on the heart are those produced by obstructed outflow from the lungs and left auricle, together with such as are usually caused by reflux into the left ventricle—namely, hypertrophy and dilatation of the left auricle and right ventricle, and in the case of the left ventricle, such a degree of hypertrophy and dilatation as would follow regurgitation of a diminished volume of blood from the aorta, diminished in consequence of the stenosed mitral opening. In one case the mitral lesion predominates, and the effects on the heart and circulation are essentially the same as in uncomplicated mitral narrowing. In another this defect is subordinate to the aortic lesion, and the secondary changes in the heart are chiefly such as are found in aortic insufficiency.

Symptoms.—The symptoms are consequently determined by the predominating lesion. In all examples of this combination there is more or less dyspnœa of effort, but when the mitral surpasses the aortic defect in gravity this symptom is more pronounced.

Thus I have observed two female patients with this combination. In one the aortic regurgitation was plainly the greater, and she was able to take a fair amount of exercise, even slow bicycle-riding, without special discomfort. If the effort became too severe it produced palpitation and breathlessness. The other woman in whom the mitral defect predominated, and was still further complicated by pericardial adhesions, showed great hepatic and considerable general venous engorgement and complained of weakness and decided shortness of breath upon even slight exertion. Both these patients broke down their compensation while under my observation, and in both this rupture proved irretrievable. The latter was given a course of baths, after having been confined to bed for a number of weeks. They failed utterly to reinstate the heart. Digitalis also proved powerless. Dropsy did not appear, but the circulation became extremely feeble, temperature remained persistently subnormal, falling on several occasions to 96° F., and once to 95° F., dyspnœa grew greater, and death took place one week after she returned to her Dakota home, under what final appearances I have not been able to learn.

The other patient considered herself in usual health until midsummer of 1901. Then, apparently as a result of the intense heat, the fatigue of a short journey, and an attack of indigestion, following a too hearty supper that same day, she began to suffer from most annoying palpitation whenever she walked about, no matter how slowly. Weakness also set in, and with the palpitation increased in spite of digitalis and other therapeutic measures. These symptoms at length obliged her to keep her bed, and even then her condition grew so much worse that she was brought back to Chicago.

I found her in a deplorable plight. The pulse was extremely small and weak, about 100, and the right appreciably smaller than the left. The right arm and a portion of the right thoracic wall were œdematous, in consequence of thrombosis of the external jugular, subclavian, and axillary veins. The liver was palpable and hard, but there was no dropsy of the ankles. The bases of the lungs were dull with fine crackling râles, and she coughed up bloody sputum. The right heart was much dilated, and the sounds and murmurs were feeble. She complained much of exhaustion, slept poorly, and passed a scanty amount of urine containing a trace of albumin.

After a time dropsy of the legs set in, and towards the close of her illness thrombosis took place in the veins of the left side of the neck, with resulting œdema of the corresponding arm. There was nothing to indicate acute endocarditis, and hence the thrombosis was probably due to coagulation of the blood from pressure and stasis. This very interesting phenomenon—i. e., venous thrombosis in cases of heart-disease—has been considered more fully under Symptoms of Chronic Endocarditis (p. 205).

Diagnosis.—The diagnosis of combined mitral stenosis and aortic regurgitation is made by the discovery of the physical signs of both lesions modified and more or less obscured by each other.

Inspection shows the apex-beat displaced to the left and downward, as in aortic incompetence, but to a less extent. If the stenosis is considerable, and has led to right-ventricle hypertrophy, there is epigastric pulsation, and there may also be visible engorgement of the superficial veins.

On *palpation* the displaced apex-beat is found to be less forcible and heaving than in pure aortic regurgitation, and there is a more or less distinct and prolonged presystolic thrill, depending on the degree of mitral constriction. The characters of the pulse are also found modified. By reason of the stenosis it is small and weak, while the aortic lesion gives it a collapsing character. In one of my patients mentioned above this was fairly well marked, while in the other it was not appreciable by the finger, the pulse being distinguished by smallness and lowness of tension. In cases in which the mitral obstruction is the dominant lesion palpation is also likely to detect more or less hepatic enlargement.

Percussion discovers increased cardiac dulness in all diameters, and is of great aid in the determination of the coexistence of these two lesions. Mitral stenosis does not cause increase of dulness to the left of the nipple; and, conversely, aortic regurgitation does not occasion increase of dulness to the right. Yet in this combined lesion dulness is increased in both these directions. Consequently, the results of percussion taken in connection with those of auscultation are of the greatest possible importance.

Auscultation.—This also furnishes valuable information, although it should never be relied upon to the exclusion of the secondary physical signs perceived by the other means of investigation. The mitral disease is shown by a characteristic presystolic murmur at the apex and by accentuation of the pulmonic second sound, the aortic insufficiency by a diastolic bruit in the aortic area or upon the sternum, and transmitted downward and to the left, while the second tone in the second right interspace is enfeebled or absent. If in doubt concerning the significance of this murmur, one should auscultate the femoral artery, since when aortic regurgitation is also present there is a sharp systolic snap, and it may be also a double murmur in this vessel. Extreme mitral stenosis in its late stages may occasion pulmonary incompetence with a diastolic bruit, and therefore auscultation of the femorals is of greatest importance in the differentiation of this insufficiency from aortic regurgitation.

Prognosis.—The prognosis depends upon the degree of the two lesions and upon which predominates; yet, on the whole, the course is likely to be that of mitral stenosis.

MITRAL REGURGITATION AND AORTIC STENOSIS

A moment's reflection will convince one of the exceeding seriousness of this combination. The obstruction to the outflow into the aorta serves to intensify the regurgitation into the auricle, because the blood flows in the direction of least resistance, which in this case is backward rather than forward. If the stenosis is extreme, it leads to great stasis and exerts all the local and constitutional effects of a most pronounced regurgitation. The heart becomes enlarged in its entirety, but the hypertrophy of the left ventricle, instead of overcoming the obstruction, serves but to intensify the force of regurgitation. The work of maintaining the circulation falls chiefly on the right ventricle, and as this is a thin-walled chamber, capable of but limited compensatory hypertrophy, it will not long be able to keep up the unequal struggle.

Symptoms are those of mitral disease of an extreme degree, and do not need to be recapitulated.

Diagnosis.—The pulse is small and feeble, while its rate and rhythm are determined by the state of compensation. The apex-beat is displaced downward and outward, and relative cardiac dulness is increased in all directions. Two systolic murmurs are audible, one in the mitral area, and one in the aortic, which are to be distinguished from each other by their different points of maximum intensity, by their propagation, and by their different quality. The former, blowing and softer, is transmitted to the left, while the aortic, lower pitched and rougher, is propagated upward into the arteries of the neck. There is intensification of the pulmonic second and diminution of the aortic second sound. The chief difficulty of diagnosis does not lie in recognising the presence of the mitral insufficiency, but in determining whether or not this is relative, in consequence of dilatation of the left ventricle secondary to the long existing aortic stenosis.

Prognosis.—Under the most favourable circumstances the prognosis is grave, since the compensation on the part of the right ventricle is likely to be short lived, and when once ruptured cannot possibly be restored. Moreover, both pulmonary and tricuspid insufficiency are likely to result when compensation fails, and then render the prognosis hopeless.

AORTIC REGURGITATION AND MITRAL REGURGITATION

This combination is not infrequently encountered in the late stages of aortic insufficiency when dilatation of the ventricle has led to relative incompetence of the auriculo-ventricular valve. It may, however, be seen as a combined lesion when both defects are the result of structural alteration. The combination is a grave one, and yet, as stated by Bacelli, a double regurgitation of the kind under discussion does not begin to be so serious as obstruction at the aortic and leakage at the mitral ostium.

Symptoms.—The influence of the mitral lesion is to lessen the effect of the aortic regurgitation on the general system, since a part of the blood intended for the aorta is diverted into the auricle, and the arterial system is not so violently distended by each blood-wave. Arterial tension does not present such a striking contrast during systole and diastole as in uncomplicated insufficiency of the semilunar valve. For this very reason, however, the arterial blood-supply to the various organs and tissues is diminished, and there is marked arterial anæmia.

In addition there are the symptoms of venous congestion, only limited by such capacity for compensatory hypertrophy as resides in the right ventricle. The heart is likely to attain enormous size, as shown by the position of the apex-beat far to the left of the nipple and downward, and by great increase of both relative and absolute cardiac dulness.

Diagnosis.—This is not usually a matter of much difficulty. The pulse is small yet collapsing, and there is increased dulness both to left and right. Auscultation reveals both a basic diastolic and apex systolic bruit, with feebleness of the aortic second accentuation of the pulmonic second, and often absence of the systolic sound at the apex. Inspection and palpation disclose passive congestion of the venous system and abdominal viscera. In case the diastolic bruit is likely to be thought a mitral diastolic one, femorals is of greatest importance in the differentiation of this insufficiency from aortic regurgitation.

Prognosis.—The prognosis depends upon the degree of the two lesions and upon which predominates; yet, on the whole, the course is likely to be that of mitral stenosis.

MITRAL REGURGITATION AND AORTIC STENOSIS

A moment's reflection will convince one of the exceeding seriousness of this combination. The obstruction to the outflow into the aorta serves to intensify the regurgitation into the auricle, because the blood flows in the direction of least resistance, which in this case is backward rather than forward. If the stenosis is extreme, it leads to great stasis and exerts all the local and constitutional effects of a most pronounced regurgitation. The heart becomes enlarged in its entirety, but the hypertrophy of the left ventricle, instead of overcoming the obstruction, serves but to intensify the force of regurgitation. The work of maintaining the circulation falls chiefly on the right ventricle, and as this is a thin-walled chamber, capable of but limited compensatory hypertrophy, it will not long be able to keep up the unequal struggle.

Symptoms are those of mitral disease of an extreme degree, and do not need to be recapitulated.

Diagnosis.—The pulse is small and feeble, while its rate and rhythm are determined by the state of compensation. The apex-beat is displaced downward and outward, and relative cardiac dulness is increased in all directions. Two systolic murmurs are audible, one in the mitral area, and one in the aortic, which are to be distinguished from each other by their different points of maximum intensity, by their propagation, and by their different quality. The former, blowing and softer, is transmitted to the left, while the aortic, lower pitched and rougher, is propagated upward into the arteries of the neck. There is intensification of the pulmonic second and diminution of the aortic second sound. The chief difficulty of diagnosis does not lie in recognising the presence of the mitral insufficiency, but in determining whether or not this is relative, in consequence of dilatation of the left ventricle secondary to the long existing aortic stenosis.

Prognosis.—Under the most favourable circumstances the prognosis is grave, since the compensation on the part of the right ventricle is likely to be short lived, and when once ruptured cannot possibly be restored. Moreover, both pulmonary and tricuspid insufficiency are likely to result when compensation fails, and then render the prognosis hopeless.

AORTIC REGURGITATION AND MITRAL REGURGITATION

This combination is not infrequently encountered in the late stages of aortic insufficiency when dilatation of the ventricle has led to relative incompetence of the auriculo-ventricular valve. It may, however, be seen as a combined lesion when both defects are the result of structural alteration. The combination is a grave one, and yet, as stated by Bacelli, a double regurgitation of the kind under discussion does not begin to be so serious as obstruction at the aortic and leakage at the mitral ostium.

Symptoms.—The influence of the mitral lesion is to lessen the effect of the aortic regurgitation on the general system, since a part of the blood intended for the aorta is diverted into the auricle, and the arterial system is not so violently distended by each blood-wave. Arterial tension does not present such a striking contrast during systole and diastole as in uncomplicated insufficiency of the semilunar valve. For this very reason, however, the arterial blood-supply to the various organs and tissues is diminished, and there is marked arterial anæmia.

In addition there are the symptoms of venous congestion, only limited by such capacity for compensatory hypertrophy as resides in the right ventricle. The heart is likely to attain enormous size, as shown by the position of the apex-beat far to the left of the nipple and downward, and by great increase of both relative and absolute cardiac dulness.

Diagnosis.—This is not usually a matter of much difficulty. The pulse is small yet collapsing, and there is increased dulness both to left and right. Auscultation reveals both a basic diastolic and apex systolic bruit, with feebleness of the aortic second accentuation of the pulmonic second, and often absence of the systolic sound at the apex. Inspection and palpation disclose passive congestion of the venous system and abdominal viscera. In case the diastolic bruit is likely to be thought a mitral diastolic one, its real nature may be ascertained by auscultation of the femoral arteries.

Prognosis.—When the combined defects are both primary, a fair degree of compensation may be attained and preserved for a time. When, however, cardiac adequacy is once seriously impaired, there is but small prospect of its restoration. If the mitral leak is secondary, it indicates such a grave loss of ventricular tone as to make practically hopeless the possibility of again closing up the mitral orifice by treatment, no matter how skilful and energetic it may be. This was shown by the history of the cases narrated in the chapter on Aortic Regurgitation.

AORTIC STENOSIS AND AORTIC REGURGITATION

This combination is not very infrequent, but does not exist so often as the diagnosis is made. This holds true particularly with regard to cases of aortic incompetence. The rough systolic murmur so commonly heard in persons who present unequivocal signs of free regurgitation through the aortic ostium leads most inexperienced auscultators to conclude that there must also be stenosis. This inference is erroneous, however, as shown by necropsies. Vegetations about the orifice, the ragged and stiff leaflets, atheromatous patches on the surface of the aortic intima, are all capable of throwing the blood-stream into audible vibrations as it passes through the ring without in the least acting as an obstruction, an important fact in its bearing on the clinical features of the case.

In predominating aortic stenosis, on the other hand, some degree of regurgitation is very likely to occur, as has been stated in the chapter dealing with obstruction at this orifice. The thickening and rigidity of the valve flaps, which prevent their being thrown widely open by the emerging stream, also interfere with their complete closure as ventricular contraction ends. Hence a portion of the blood-wave finds its way back into the ventricle. In other cases one of the cusps may be fenestrated, or for some other reason incompetent, whereas its fellows are not, being only incapable of opening in a normal manner.

Symptoms.—The symptoms produced by combined aortic stenosis and regurgitation partake in character and gravity of the features which are special to the predominating lesion. If incompetence is the greater, compensation is possible for years without the individual being made aware of its presence. If stenosis predominates and is pronounced, the left ventricle is not likely to establish such a degree of hypertrophy as will maintain complete adequacy for very long. The reflux, even if slight, as measured by actual quantity, is yet sufficient to cause more or less dilatation of the chamber, and hence the driving force of its wall is impaired. Consequently, the patient is more apt to notice some breathlessness and perhaps palpitation under conditions that ought not to affect him were either stenosis alone or regurgitation alone the lesion.

Physical Signs.—The physical signs are modified also by this combination, and display in varying proportion the characters of each defect. Thus the pulse is neither so large and collapsing as in pure aortic regurgitation, nor so small and slow as in uncomplicated stenosis, but is collapsing and also small. Capillary pulsation and Duroziez's double femoral bruit are either absent or very imperfectly obtained.

The impulse of the heart against the chest-wall is not so forcible and extensive as in free regurgitation, and the apex-beat in size and displacement partakes rather of the character of stenosis. Hypertrophy of the left ventricle is more apparent than is its dilatation with thickening.

The hand is very apt to perceive a systolic thrill in the aortic area, and percussion demonstrates that the heart is not so large as in uncombined aortic insufficiency.

There are two murmurs, of which the systolic is likely to be intense and rasping, while the diastolic is of inferior prominence in all respects. The sounds normally heard in the second right interspace and in the cervical arteries are likely to be absent and replaced by murmurs.

Diagnosis.—The diagnosis of this combination is as a matter of fact very difficult, and it is often impossible to determine definitely whether both conditions are united or not. This is emphatically true if the case is seen for the first time after compensation has failed. Relative mitral insufficiency or pronounced feebleness of the left ventricle may then modify the pulse, sounds, and murmurs in the manner just described. However, if the femoral artery is auscultated, and the left side of the heart is accurately outlined by percussion, Duroziez's sign will declare the freedom of the reflux, and percussion will demonstrate the enormous enlargement of the left ventricle secondary to free regurgitation without obstruction.

A moderately slow, small, yet collapsing pulse, a vigorous, rather circumscribed, not greatly displaced apex-impulse, a systolic aortic thrill and bruit without powerfully throbbing and thrilling cervical arteries, absence of double femoral souffle, and no demonstrable capillary pulse—these signs, together with a regurgitant murmur, would justify the conclusion that stenosis and insufficiency coexist, but that the former probably predominates. The sphygmograph ought to show the rounded summit and anacrotic notch of obstruction and the ill-sustained down stroke of regurgitation (see Figs. 54 and 66).

Prognosis.—The prognosis of this double defect is certainly far from favourable, either as to length of life or as to restoration of heart-power, when this has once given way. This certainly applies to pronounced stenosis with regurgitation, whereas it is conceivable that a minor degree of narrowing might, by rendering regurgitation less free, serve to protect the wall of the left ventricle against the speedily damaging effects of free reflux through a widely patent orifice.

CHAPTER XV

THE PROGNOSIS OF VALVULAR HEART-DISEASE IN GENERAL

Something has been said already on the subject of prognosis in the chapters devoted to the individual valve-lesions, and therefore some repetition will be unavoidable. In attempting to forecast the course and termination of a given case one should consider (1) the special characters of the lesion, (2) the degree of the secondary effects in the heart and other organs, and (3) extraneous factors of age, temperament, environment, etc.

The characters of valvular disease which influence prognosis are its nature, location, and degree, and these cannot always be considered separately. As a general proposition, it may be stated that stenosis is a more serious defect than is regurgitation, and yet its gravity depends largely on its location. Furthermore, the amount of disturbance to the circulation is determined so much by the degree of the local defect that this latter may render most serious a valvular disease, which from its nature and situation alone would ordinarily furnish a more favourable prognosis. In fact the forecast is so largely based on the conditions of each case that one would go far astray if he were to be guided by general principles alone.

Although aortic insufficiency is to be ranked first as regards gravity, still a distinction should be made between cases originating in the young in endocarditis, commonly rheumatic, and those of atheromatous origin, observed at or beyond middle age. In the former group great compensatory hypertrophy and a healthy heart-muscle may enable the organ to functionate adequately for many years, far longer indeed than do many cases of mitral disease, although in itself this latter is considered a less serious lesion. On the other hand, when aortic incompetence is due to a sclerotic process, the myocardium is rarely healthy and compensation is short lived, or indeed is never perfect. In such a case prognosis is grave from the start. It is in this particular lesion that sudden and unexpected death is likely to take place. Indeed, it is almost the only valvular disease which so terminates, since when death occurs unexpectedly in other defects it is very exceptionally instantaneous, and then is the result of some accident, such as embolism, or it terminates weary weeks or months of failing heart-power.

In aortic regurgitation it is not very rare for patients to fall dead unexpectedly in the midst of apparently good health. Whenever compensation shows unmistakable signs of failure, sudden death in this disease is not a very remote possibility. Moreover, compensation may be broken at any time by a rheumatic attack, and once impaired it is rarely restored. Absence of the aortic second sound and dilatation of the left ventricle are therefore prognostically grave, since they indicate free reflux and feeble ventricular resistance.

Stenosis of the aortic ring presents a less grave prognosis than does regurgitation at this orifice. The reason for this difference is to be found in the effect of the two lesions on the wall of the left ventricle. A narrowing of the outlet leads to hypertrophy with relatively little dilatation, unless of course the obstruction be so pronounced that the chamber is unable to empty itself during systole, and stasis results behind the point of constriction. So long as the hypertrophied ventricle is able to discharge its contents with each contraction, and the effect of the lesion is limited to the ventricular wall, the prospect of a continuance of life for many years without distressing symptoms, and even of death at the end through some intercurrent affection, is good. When, however, compensation in this disease is once destroyed, there is small likelihood of its repair, and the prognosis becomes very serious. Yet in this, as other lesions, it is its severity, even more than its nature and location, which determines the degree of its seriousness. An extreme stenosis as regards length of life is even worse than free regurgitation. When the two lesions are combined the prognosis is as a rule more unfavourable.

Of the two mitral defects, it is generally conceded that *stenosis* is the more serious. One reason for this is that the disease is not stationary, but tends to grow more pronounced in consequence of contraction of the newly formed fibrous tissue and of the increase of fibrine deposited upon the vegetations. Another reason, as we shall see later on, lies in the greater intensity of the secondary effects on the heart. Mitral regurgitation, on the other hand, is under ordinary circumstances the most favourable of the four lesions situated in the left heart. When the leak is not too free and there are no serious complications, such as aortic stenosis and adherent pericardium, the defect in question is not incompatible with long life and great mental and bodily vigour. It is possible, however, for the regurgitation to be so free that this relatively benign disease is thereby converted into a very serious one. Leyden states that sudden death occurs in only 2 per cent of mitral disease.

With the exception of relative tricuspid insufficiency, diseases of valves of the right heart are so infrequent that nothing needs to be added to what has been said already concerning their prognosis in the respective chapters. Incompetence of the right auriculo-ventricular valves secondary to other diseases is generally regarded as of serious import, not because it threatens life directly, having, as it is said, a safety-valve action, but because it indicates serious disproportion between the degree of the primary disease and the strength of the right ventricle. If it occurs with anything like the frequency claimed for it by Gibson, then one should not attach to it a very unfavourable prognosis. Nevertheless the degree of importance to be attributed to it depends much on the nature of the primary affection. If it be secondary to vesicular emphysema or to valvular disease of the left side of the heart, as pronounced mitral stenosis, the development of tricuspid regurgitation must be looked upon as an omen of impending disaster. This form of tricuspid disease cannot be regarded as a separate and independent affection, and therefore should be classed among the secondary effects of valvular disease, which are now to be discussed in their bearing on prognosis.

From the foregoing it is evident that although the nature and seat of valvular defects influence their prognosis, yet it is their *intensity* to which we must chiefly look when directing our attention to the heart. It has been distinctly stated in previous chapters that in estimating the extent of a valvular defect one must not rely upon the intensity of the murmur, but upon the evidences of disordered circulation. These are the secondary effects or signs which are of such value oftentimes in making a diagnosis as well as in stating the prognosis.

One reason for the grave outlook in cases of mitral stenosis is the fact that this defect occasions widespread stasis in the vessels of the pulmonary and venous systems, while the diminished supply of blood to the left ventricle leads to shrinkage in the size of this cavity. If the left auriculo-ventricular opening has become greatly reduced in diameter, no amount of vigour of the left auricle and right ventricle can maintain the equilibrium of the blood-stream. It is only a matter of time when the pulmonary system and right heart, systemic veins, and abdominal organs are bound to become engorged.

In mitral incompetence, on the other hand, the left auricle and pulmonary veins may be able to bear the brunt of the regurgitating stream for a long time. Moreover, the left ventricle undergoes hypertrophy, and forcibly ejects into the aorta all that portion of the blood that does not escape into the auricle. There is not so marked a tendency to disturbance of general nutrition. Yet, of two typical cases of mitral disease, the one constrictive and the other regurgitant, if the former with its natural tendency to greater stasis actually displays less pronounced secondary effects, it offers a better rather than a graver prognosis, because compensation is complete. The general venous stasis in the regurgitant case evinces either such a freedom of reflux that the parts behind could not long withstand it, and compensation was necessarily lost, or that compensation was not able to take place at all. Even if treatment should succeed in reinstating the circulation, still the fact of compensation having once been lost would render the prognosis worse than it would be in the case of stenosis in which compensation had never been impaired.

Again, compare a case of perfectly compensated insufficiency of the aortic valves with one of extreme narrowing of that orifice in which dilatation of the left ventricle is beginning to outbalance the hypertrophy, and signs of stasis are appearing in the pulmonary and general venous systems. In one, secondary effects are limited to the heart and shown by the adjustment of the left ventricle to the altered conditions. In the other they have passed beyond the heart and invaded the remainder of the circulatory apparatus. It is plain that here the degree of the lesion has reversed the usual order of things as respects the prognosis in these two valvular defects.

The foregoing remarks show how unreliable would be a prognosis in valvular heart-disease, which was not based on a careful study of the extent, even more than the nature and location, of the particular defect, and that individual cardiac conditions determine the relative gravity of each case. Nevertheless, I must repeat that my experience leads me to agree with Broadbent in the opinion that, generally speaking, aortic regurgitation is the most serious and mitral regurgitation the most favourable of the four valvular diseases of the left heart. The two stenoses occupy an intermediate position, and of these, mitral constriction is the graver. This subject is still further complicated by the consideration that there are still other factors that must be reckoned with. For the most part these are of minor importance, and yet some of them make strongly for or against an encouraging forecast.

Complications.—The gravity of any valvular defect is necessarily enhanced by the existence of complications, although to what extent is determined in great measure by the nature of the complication. Intercurrent acute disorders, which act as complications while they last, are considered by themselves. Here are discussed only such chronic local alterations and diseases of other viscera as must of a necessity unfavourably affect the course of valvular lesions. Pericardial adhesions, whether strictly internal or such as bind the heart to some of the surrounding parts, certainly exercise a malign influence, since they interfere more or less seriously either with the establishment or the maintenance of adequate compensation. Their effect is specially detrimental if by fixation of a chamber in the state of dilatation they prevent its reduction and efficient hypertrophy. I have seen this more than once exhibited in a case of mitral incompetence in which fixation of the left heart threw extra strain upon the right ventricle, as evinced by its ready dilatability. When a chronic adhesive mediastinitis holds the right heart adherent back-pressure on the two cavæ and liver is increased. The pseudo-cirrhosis of the liver leads in time to obstinate ascites, and patients succumb to the hepatic complication long before they would be likely to die from cardiac inadequacy alone. Moreover, an adherent pericardium not infrequently renders futile therapeutic efforts which prove highly efficacious in cases without such complication.

The association of two or more valve-lesions affects prognosis, not by shortening life necessarily, although such is likely to be the effect, but by rendering impossible the development of perfect compensation and compelling extraordinary carefulness, lest what small measure of compensation already exists be broken down altogether. The reader will find more on this subject in the chapter on Combined Valvular Lesions.

It goes without saying that *chronic nephritis* is a very grave complication. Not only does the renal act badly on the cardiac affection, but this latter, by lowering blood-pressure in the renal arteries, intensifies the insufficiency of the kidneys. The evils of uræmia are then likely to be added to those of defective circulation. The chronic nephritis renders it unlikely that the patient will live out the term of years that would naturally be granted him by his valvular defect alone. The kidney complication also renders less availing all attempts to remove dropsy whenever it appears.

Pulmonary tuberculosis is not often seen in combination with valvular disease, excepting of course pulmonary stenosis. When it occurs, however, I believe it enhances the gravity of prognosis, for I cannot see how they can fail to react injuriously on each other. Anything which, like valvular disease, impairs nutrition must necessarily lessen the likelihood of successful resistance to tuberculosis, while the destruction of lung-tissue must seriously affect the already damaged heart.

Harmful blood-states, as *chlorosis* and *anæmia*, affect prognosis in proportion to their severity and their amenability to treatment.

Rheumatic Diathesis.—Some individuals display a marked tendency to rheumatic attacks, either acute or subacute, and every now and then suffer from pains in shoulder, wrist, or other joints. In such the outlook is not bright, for the reason that any one or all of these mild attacks may be attended by fresh endocardial inflammation either of the same or other valves, or that pericarditis may develop. Even if an active endocarditis is not excited, the changes already set up in the valves may be rendered progressive. Consequently, a case furnishing favourable prognosis originally may be converted into one of a most serious nature. In a word, therefore, recurrences of rheumatism, no matter how mild, are to be regarded as affording a gloomy prognosis in any case of valvular disease.

Digestive and Bronchial Disorders.—These, like rheumatism, yet in a different way, are capable of unfavourably affecting prognosis. Disturbance of the digestive function is not infrequently observed in victims of valvular disease in whom careful examination fails to detect signs of secondary effects in other organs. The chylopoietic viscera may have their function impaired by lack of arterial blood of good quality, cardiopaths being often anæmic, or in aortic cases by a defective flushing with arterial blood, or in mitral patients by passive congestion, this last being too slight to be recognised by ordinary means of examination. Whether the indigestion is owing to such causes or is the result of improper food or faulty habits in eating, it is likely to impair general, and hence cardiac nutrition, and thus render prognosis less encouraging.

A tendency to acute bronchial catarrhs in mitral patients not only evinces greater pulmonary congestion than is otherwise apparent, but also renders them liable to an attack of bronchitis, which may at any time severely strain compensation. In them, therefore, prognosis cannot be looked upon as so favourable as if they were less sensitive to atmospheric changes and did not so easily get up a cough, for the severe expiratory effort of coughing subjects the right ventricle to added strain.

Age.—The prognosis of valvular disease is more serious at either extreme of life and most favourable in young adults. In elderly individuals the myocardium is apt to be more or less degenerated, and although, as Leyden believes, compensation is often as perfect as in the young, it is more easily destroyed. Furthermore, the sclerotic process, which is usually responsible for the valvular defect, is progressive, and one possesses no means of forecasting whether these changes will progress slowly or rapidly. I recall the instance of a gentleman of sixty-four in whom I detected signs of aortic sclerosis and probable coronary sclerosis in explanation of his attacks of angina without any evidence of valvular incompetence or of stenosis. Yet at his death, less than three years subsequently, the autopsy disclosed, I have been informed, well-marked insufficiency of the atheromatous aortic valves, signs of the lesion having developed and been detected by his physician some months prior to his death.

The gravity of the prognosis in childhood is attributable to a variety of causes. In the first place, the heart-muscle, although free from degenerations, is easily exhausted. The chest is small and affords scant room for the often enormous hearts observed in children with long-standing valvular disease. Any one who has seen much of valvular disease in children must have observed that they are strikingly unconscious of symptoms which in adults occasion complaint. They are highly sensitive to pain, yet appear to pay no attention to palpitation and shortness of breath during play; although the onlooker may observe tumultuous action of the heart, hurried breathing, and cyanosis. Children are therefore very likely to overstrain their already damaged hearts; and that this does not occur more frequently is quite remarkable. These little ones are excitable and emotional, and therefore unable to exert the self-control so often necessary for the preservation of compensation. They often display astonishingly vigorous appetites and overload their stomachs with the sweetmeats and dainties they crave, and are permitted by indulgent parents to have. These ferment with the formation of gas, which, distending the digestive organs, causes them to crowd upward upon and still further embarrass the heart in its action. Lastly, rheumatism in childhood is so insidious and atypical that it is very frequently overlooked. Prompt and efficient treatment is not instituted, and this disease being frequent in early years of life excites fresh attacks of endocarditis, lights up a pericarditis, or renders existing valve-lesions progressive. Mitral stenosis in young children is particularly unfavourable. It may be briefly stated that valvular disease at this period of life is very likely to end fatally before the patient reaches adult age either directly or through complications.

Temperament.—This possesses a not unimportant relation to the prognosis of the diseases now under consideration. The patient who is impulsive, impetuous, and thoughtless is like a child unaccustomed to self-control, and if required to exercise self-restraint frets and chafes in spirit. Such a person will be forever committing indiscreet acts, and will only acquire with difficulty that patience and equipoise of spirit which serve as ballast to damaged hearts. Individuals given to outbursts of anger, to worry, to fretting over trifles, and who appear never to become reconciled to their physical disability, can never be expected to retain their compensation so well or so long as will those who always have themselves well in hand. Verily, all cardiopaths should bear in mind that Bible utterance, " He that ruleth his own spirit is greater than he that taketh a city."

Sex.—Mitral disease, and in particular mitral stenosis, is more frequent in the fair sex, while men are more subject to aortic insufficiency. In a sense, therefore, sex may be said to exert a general influence upon prognosis. The inquiry that now concerns us is how does sex affect the prognosis of a given valvular lesion after it has once been established, without reference to its nature. In other words, what is the relative prognosis, *seteris paribus*, of the same defect in the two sexes. This is a very difficult matter for decision, since it involves questions of habits, occupation, etc. Females are exposed to certain perils of pregnancy and child-bearing, while, on the other hand, men have to encounter even greater dangers incident to occupations that often produce cardiac overstrain. The reader will find these influences discussed at some length in the chapter on Treatment of Valvular Disease in General. One respect wherein women usually furnish a more favourable prognosis than do males is that of habits—that is, a greater freedom from the injurious effects of excess in tobacco, alcohol, and venery. Women are generally held to be more emotional and excitable than men, yet in the matter of self-control they seem to me to possess an advantage over their brothers. The most marked instances I have ever seen of apprehension—nay, of alarm and nervous agitation—lest the examination result in the discovery of a heart-lesion, have been in young men. The female sex is more prone to anæmia and chlorosis, and the injurious influence of these blood-states is too well known to require more than this passing reference. In most other respects I think the question of sex resolves itself into that of the individual.

Occupation.—This exerts a powerful influence upon prognosis. The day labourer who earns his daily bread by the sweat of his brow cannot be expected to keep his compensation intact for so long as will he whose vocation does not subject his heart to the possibility of overstrain. All authors are agreed in the declaration that nothing in the daily life of these patients affects their hearts, and hence the prognosis, more disastrously than does severe and prolonged or too oft-repeated physical exertion. This is particularly true of mitral narrowing, even in the stage of compensation. Patients with well-compensated insufficiency of the aortic valves may endure overstrain for a time without apparent injury; but so soon as dilatation of the left ventricle has begun to gain the ascendency over hypertrophy, a continuance of the strain will inevitably result in a breakdown, and that too at no very distant date in most instances.

Habits.—These are matters of utmost importance if the lives of patients with valvular disease are to be prolonged. They should be minutely inquired into, therefore, by the medical attendant. The daily life of these sufferers should be ordered on the principle of moderation in all things. Whatever is injurious to a healthy person is doubly so to one with an unsound heart. Consequently a prognosis which, as regards everything else, may be good, may be rendered very uncertain, if not actually bad, by the discovery of evil practices. By these are meant particularly excess in tobacco, alcohol, or other narcotics, and in sexual indulgence. But patients may also increase the gravity of prognosis by gluttony, loss of sleep, exciting novel reading, gaming, etc.—in short, by whatever promotes nervous and cardiac excitement.

Home Surroundings.—These include all those matters of sanitation, as dampness, sunshine, ventilation, drainage, the ability to obtain suitable food and clothing, freedom or not from domestic worry and annoyances, opportunity for recreation, etc.—in a word, the residential and social conditions which in all of us make for happy, contented lives.

The prognosis in the case of the poor man cannot be expected to be as good as that of him who is able to command everything that can minister to his comfort and well-being. If, e. g., a patient with mitral stenosis or a failing aortic insufficiency is compelled by the exigencies of his purse or environment to labour or to ascend wearisome flights of stairs or steep acclivities upon returning to his home, no matter how often this may be, he can hardly be expected to keep this up without eventually suffering injury. These and many other matters may seem too obvious to require mention, and yet they are details which the physician must take into consideration if he would form a reliable prognosis.

The Probable Effect on the Patient of the Knowledge of his Lesion.—This is a matter, in my opinion, having a decided bearing on prognosis, and which, therefore, should be discussed. Physicians and the laity generally believe a cardiopath must not be informed of the fact when he is found to have a cardiac defect, lest he be alarmed and become morbid and introspective. Doubtless there are many nervous, apprehensive persons who would be harmfully affected by such knowledge. When such is the case I believe it renders prognosis less favourable, because if kept in ignorance of his true condition he is not prepared to avoid whatever may be harmful. If detrimental influences are to be shunned, patients must have explained to them how and why these are injurious to them, since the doctor's dictum in this regard is not enough for an intelligent person. Kept in ignorance or put off with an evasive answer, he may be set to pondering and conjecturing, and hence to fancying his condition is worse than it really is. I believe, therefore, that it is a positive gain to a cardiopath to acquaint him with at least a part if not all of the truth. Of course he does not need to be informed with brutal abruptness, but gently and in a manner calculated not to frighten him unduly. The individual who cannot bear even a part of the truth without detriment will assuredly furnish a less favourable prognosis than he who, knowing the truth, accepts it philosophically, and determines to make the best of a bad bargain.

The Effect of Digitalis on the Patient.—It goes without saying that when valvular disease has reached such a stage as to necessitate the administration of digitalis the prognosis is not good even at the best. Thus much any one knows, but only a few, if any, are able to prognosticate how much longer the heart is going to bear up, even sustained by such a prop. In such a case, as pointed out by Leyden, a certain degree of information may be derived from a study of the effect of the remedy.

If the beneficial action of digitalis is quickly lost after its administration has been discontinued, and the heart manifest its need of this tonic by a speedy return of symptoms, the prognosis is serious, for it indicates myocardial inadequacy. It is a still more unfavourable indication if from time to time the dose of digitalis has to be increased to maintain its effect, for it points to the not distant arrival of a time when the heart will cease to respond to the remedy, and the end will not be far to seek.

The Relation of Prognosis to Life Insurance.—There was a time when an individual with valvular disease was rejected indiscriminately by all insurance companies. In England, and by some companies now in this country, some of these patients are accepted as "defective risks," and therefore it is in order to discuss this subject in this place. There are two classes of persons with valve-lesions whom I would reject except possibly for a very limited term of years, and only then at so high a premium as to make it almost prohibitive. These are cases of pronounced mitral stenosis and insufficiency of the aortic leaflets. Even when the latter appears compensated there is always the possibility of sudden and unexpected death, which, as already stated, renders prognosis very uncertain. Stenosis of the left auriculo-ventricular orifice is progressive, and how rapidly this tendency will declare itself no one can foresee. On the contrary, mitral regurgitation, and to a somewhat less degree aortic stenosis, may sometimes be considered reasonably safe risks as defectives. It will be noted that I say sometimes. This is because, no matter how excellently the lesion may be compensated, there are circumstances of individuality or environment which determine prognosis adversely. Therefore the examiner should consider exhaustively and intelligently all those factors which have a bearing directly or remotely on the prospect of the patient living as long as the characters of his disease might be expected to allow. Laborious occupations and bad habits are, in my opinion, a bar to safe insurance of these risks. On the other hand, a robust young man who knows that his mitral valves leak, and who is determined to order his daily conduct in a manner calculated to afford his heart the very best chance of carrying him through to middle or advanced age, may often prove a safer risk than many another sound man who banks on his fine health and splendid physique.

CHAPTER XVI

THE TREATMENT OF VALVULAR HEART-DISEASE

From a therapeutic standpoint, cases of valvular disease are to be divided into three classes, according to the state of compensation: (1) Those in which the lesion is compensated, (2) in which compensation is incomplete, and (3) in which cardiac inadequacy is so pronounced that compensation is wholly wanting. We call a valvular defect compensated when the cardiac pump, in spite of its defect, is able to maintain the circulation in nearly or quite its normal state, and there are no symptoms to make the patient aware of his malady. Under such circumstances laborious occupations, athletic exercises, and games or outdoor sports requiring considerable strength and agility are endured without more breathlessness or palpitation than are usual with persons having sound hearts. In the second class, patients are still able to perform their daily duties and engage in some of the less severe sports, but it is with more or less distress and evident signs of heart-strain. There are different degrees of imperfect compensation in this class, and hence it is one of wide limits. In the third class, in which compensation is wholly lost, patients are not only incapacitated for physical exercise, but the circulatory disturbance is shown by stasis, generally by œdema, and by subjective symptoms that are present even when the patient is at rest. When compensation is perfect, examination of the heart discloses the existence of a lesion, but no secondary effects in the general circulation. In the second class signs of more or less visceral and venous congestion are detected, although subjective symptoms may be insignificant, and in the third these reach their severest grade. It is evident, therefore, that treatment appropriate to the last stage is not indicated in the first. Neither do patients whose compensation is still maintained intact require the same strict management as do those who are beginning to manifest failing heart-power. Consequently, in dealing with the management of valvular heart-disease, I shall consider it with reference to the three divisions just made.

I. COMPENSATION BEING STILL PERFECT

The object of management in this stage is the maintenance of cardiac power. Occasionally a patient with valvular disease seeks medical advice for the purpose of learning how he can preserve his heart in *statu quo.* As a rule, however, such a compensated lesion is discovered accidentally by the physician, who is then confronted by the query whether in case the patient is ignorant of his heart-disease he should be informed of it or not. I hold that in such a case the answer must depend upon the circumstances of the case, such as the temperament of the individual, his habits, and the nature of his employment and manner of life. If the knowledge that he has heart-disease is likely to frighten him and render him introspective, then the knowledge would better be withheld, unless, of course, he is leading a kind of existence calculated to break down his compensation. Under such circumstances it may be necessary, and the part of wisdom, to inform him that his heart is not sound, and is likely to be damaged by his manner of living. In such an instance, however, the information should be imparted in a manner not calculated to create needless alarm. If the individual is reasonable and cool-headed, particularly if his pursuits are active, I believe he should be plainly told of the existence of his valvular defect, for, other things being equal, the knowledge by a person that he has a *locus minoris resistentiæ* is likely to make for a longer lease of life.

Since, then, the aim of management in this stage is to preserve compensation, the physician must concern himself with the minutest details of the patient's daily life. He would take a narrow view of a case indeed who contented himself with the question of medicinal treatment. *Compensated valve-defects require not drugs, but instruction upon the following points:*

Exercise.—It may be laid down as a general proposition suitable to all forms of compensated valve-defects that when any kind of exercise does not produce symptoms of cardiac strain it may be permitted. Indeed, as will be seen later on, judicious exercise promotes compensatory hypertrophy in some forms of valvular disease. There are other lesions, however, which by their very nature are theoretically likely to and often actually do suffer injury in time from severe bodily exertion. This statement applies particularly to cases of mitral stenosis. If the left auriculo-ventricular opening is but slightly constricted, considerable, even severe physical effort may be endured without symptoms, but as a rule some shortness of breath is experienced, and patients should be explicitly warned against persisting in their exertion when dyspnœa is felt. The effect of muscular contraction and deepened respiration incident to exercise is acceleration of the flow of venous blood to the right heart and lungs. If the blood cannot readily pass the mitral ring, it becomes dammed back in the left auricle and pulmonic veins, engorging and overstraining the right ventricle. This may resist the stress for a time, but if the strain is too prolonged or too frequently repeated the cardiac walls finally yield and the hypertrophy, upon which adequate compensation depends, is superseded by dilatation. Therefore, patients with pronounced mitral stenosis, even when compensated, should be cautioned against violent, prolonged, or too oft-repeated exercise of a severe kind. Hurrying up stairs or hills, running, and even very rapid walking, fast bicycle-riding, sports and games that necessitate running and springing without frequent pauses to permit recovery of breath—*e. g.*, furious sparring, wrestling and fencing, lawn-tennis, basketball, and the like—are among the kinds of exercise particularly likely to harm patients with mitral stenosis, even when compensated. On the other hand, if they indulge moderately, they may enjoy rowing, paddling, and bowling. Billiards, golf, and croquet are specially suited to them, while some may be permitted to hunt, and nearly all to fish. When the constriction is not pronounced, gentle horseback riding, slow bicycling, and even the lighter kinds of gymnasium work are permissible. In specifying the kind of exercise and sport to be allowed, the physician should always bear in mind the personal equation. The degree of the lesion and the gravity of its secondary effects, even more than the nature of the lesion, determine the patient's ability to endure exercise without harm. The individual temperament, judgment, and power of self-restraint are also of great importance. The physician must endeavour to inform himself as accurately as possible regarding the effect of any given kind of exercise on the particular patient before coming to a decision.

What has been said of mitral stenosis applies also to cases of aortic obstruction when this is pronounced. Slight narrowing of this orifice is often compatible with great muscular exertion and active exercise. When, however, compensation is once broken in these cases, it is repaired with difficulty, if indeed at all, and therefore good judgment and careful study of each case are essential to a wise decision. In these cases exercise should not be carried to the production of palpitation, particularly prolonged palpitation. The wall of the left ventricle is susceptible of far greater hypertrophy than is that of the right; besides the effect of an aortic stenosis is confined for a time at least to the ventricle, and does not embrace the thin-walled auricle, and consequently exercise is likely to be better endured than when the obstruction is at the mitral opening.

The next in order on an ascending scale, as regards its ability to withstand the effects of exercise, is mitral regurgitation. In this disease, owing to the circumstance that during diastole there is no impediment to the filling of the ventricle, and notwithstanding that a portion of the blood gushes back during systole into the auricle, there is not the same degree of engorgement in the parts back of the seat of lesion as in mitral constriction. Of course the measure of the heart's resistance is governed by the degree of the incompetence and, as in all valve-lesions, by the state of the heart-muscle. If the leak is very free, compensation is not so apt to be complete as when the regurgitation is insignificant. In cases of well-compensated insufficiency of the mitral valve continued and severe exercise may often be indulged in without the production of annoying symptoms. This statement applies to the rheumatic rather than the atheromatous form of the lesion. I know an attorney who, fifteen years ago, when a growing lad, had a pronounced though perfectly compensated mitral insufficiency, and who played lawn-tennis enthusiastically without any other discomfort on the part of his heart than the consciousness of rapid, strong beating of the organ. Despite frequent injunctions to the contrary, he continued to indulge in this sport during several years, and is now reported to be so well that he does not know he has a heart. Another of my patients, a merchant past thirty, with a mitral incompetence in a state of admirable compensation, is much given to sparring and broadsword practice, which, he declares, never gave him any shortness of breath and only a rapid heart-action, that subsided so soon as the exertion ceased. Such instances are not rare, and yet severe physical efforts are not without danger to these patients.

Such as are fond of manly sports should be advised of the possibility of cardiac overstrain, and told to desist when excessive palpitation or pronounced dyspnœa is experienced. They feel the better for a certain amount of outdoor exercise, and when young and vigorous in other respects their heart-muscle, like their skeletal muscles, is likely to grow soft and weak if debarred from athletic sports altogether. Other things being equal, the state of the voluntary muscles is a fair index to the condition of the cardiac muscle. Of two individuals with well-compensated mitral leakage, one with well-knit muscles trained to exercise, the other unaccustomed to outdoor sports because of sedentary pursuits, the latter may break down his compensation by some effort which would be no more than child's play to the former. It is probable that persons with mitral regurgitation would be more likely to suffer injury from long running than by games that necessitate intermittent and short spurts of speed or strength. Mountain-climbing, boat-racing, and other forms of contest or strength, which experience has shown cause dilatation of healthy hearts, will bring about overstrain of diseased ones more readily and surely.

As a general rule cases of aortic regurgitation should be placed at the top of the list as regards endurance of exercise without injury. This statement does not apply to persons who have acquired their aortic incompetence after the age of forty, and therefore probably as a part or manifestation of a sclerotic process that may have invaded the myocardium also. In such, even when compensation is still maintained, exercise should always be moderate. The salvation of patients with this lesion depends on hypertrophy of the left ventricle. The great Stokes recognised this fact, and accordingly used to recommend active physical exercise to patients with this form of valve-disease. Von Ziemssen has stated that upon one occasion, when visiting Stokes in Dublin, the latter directed his attention to a man running along the street behind his wagon, and said that he was one of his patients with aortic insufficiency who was carrying out this kind of exercise at his (Stokes's) advice, for the purpose of developing left ventricle hypertrophy.

Another of Stokes's patients with the same lesion was a farmer who was able to do a day's ploughing as well as if his heart were sound, and in fact declared he felt the better for the exercise. This patient died of acute pericarditis soon after von Ziemssen learned the facts of his case, and his heart was given to von Ziemssen, who declared it weighed several pounds and was the most marked example of *cor bovinum* he had ever seen.

A well-compensated aortic regurgitation will endure arduous physical labour and the most energetic kinds of exercise so long as the myocardium is healthy. Therefore it is young or comparatively young patients whose aortic-valve disease is of rheumatic origin who are able to endure great physical exertion for many years without a breakdown. Such patients are far more likely to be unconscious of the existence of their cardiac mischief than are the subjects of mitral disease. In the former cardiac stress is manifested not so much by dyspnœa as by palpitation, and if their valve-defect dates from an attack of rheumatic endocarditis in childhood, as is often the case, they have grown up so accustomed to these palpitations that they are apt to speak of them as but a manifestation of strong action of the heart. While such patients can be quite safely permitted considerable latitude in the matter of exercise, they should nevertheless be closely watched for the first evidence of failing compensation. For so soon as the heart begins to waver in its work, bounds must be set to their activity.

Whatever is the nature of the valvular disease, the physician should always remember that after the fourth decade of life arterial degeneration is frequent, and the myocardium is likely to have suffered changes depending thereon. Consequently, liberty in exercise is more hazardous after than before this period of life, even in the case of old-standing lesions that have not previously interfered with active habits. From this time onward increasing caution must be observed, and heed given to what may appear to be but trivial symptoms of heart-strain. Should a valve become defective at this period of life, either as a result of endocarditis or atheroma, severe exercise and incautious efforts of all sorts are to be forbidden, even though good compensation seems to have been established. Every physician of experience is aware of the readiness with which compensation fails after middle age. Fortunately for such persons they have arrived at years of discretion, and find less difficulty in restraining their impulses to overdo than is the case with the young. Exercise must now be had by walking, driving, easy riding, billiards, and golf. Pulley weights, the Whitley exerciser, clubs, dumb-bells, etc., if permitted at all, are to be used under the supervision of the medical attendant or of a nurse capable of detecting signs of danger. Moderation in all things must now be the motto of these patients.

Occupation.—The principles underlying the matter of exercise should also determine the selection of a suitable occupation or the decision whether or not the patient's vocation is to be continued. The following employments are suitable for persons with mitral stenosis: Book-keeping, stenography, banking in any capacity, or other forms of desk-work, telegraphy, clerking, engraving, watchmaking, tailoring, shoemaking, harness-making, saddlery, etc., and for females the various kinds of needlework, typewriting, stenography, and desk-work. Employments that necessitate heavy lifting and the carrying of heavy parcels, as porterage, running up and down stairs, swinging of heavy hammers, etc., are injurious, since they put added strain on the right ventricle. Dusty occupations induce catarrhs and coughs, and in this respect favour bronchial congestion, to which mitral patients are predisposed already. For the same reason they should not follow occupations which expose them to vicissitudes of weather and sudden changes of temperature. Of the professions, journalism, dentistry, architecture, designing, and the various branches of art work, are all suitable—while theology, law, and such other vocations as require public speaking put a strain on the right heart and are less eligible. Teaching is not so bad, whereas the profession of medicine, especially a general practice, is too arduous and involves too much exposure for persons with pronounced although compensated mitral narrowing. A specialty permitting office practice is not so objectionable, and yet any one with this lesion should be discouraged from studying medicine, or becoming a trained nurse or masseur.

Work with light tools, as carpentering, joinery, house-painting and decorating, and even light gardening, may answer for some cases of aortic disease, when not admissible for severe mitral stenosis. Most of the occupations followed by females are not too severe for mitral patients who have good compensation, excepting such work as requires the carrying of trays heavily loaded with dishes and the frequent running upstairs. A general classification or division of occupations may be made as follows. Those that are indoors and require the use of the mental faculties rather than the muscles are suitable to mitral patients and persons with serious aortic lesions. Outdoor employments and work performed by the muscles rather than the brain may be endured by subjects with compensated aortic regurgitation and the slighter forms of aortic stenosis and some cases of mitral insufficiency. Finally, vocations attended with much excitement or nerve-strain, as locomotive driving, operating on the stock-exchange, detective and police work (these last two for other reasons as well), sea-faring, and soldiering, are unsuited to any form of valve-disease, no matter how excellent the compensation. The medical attendant can do much good by directing the choice of occupations for the young, and in the case of those who have developed disease after their work in life has been fixed by pointing out how the evils of the occupation may be minimized.

Habits.—It has already been stated that moderation should be the governing principle in the lives of cardiac patients. Excess of every kind, particularly in sexual indulgence, is to them most injurious. It not only occasions a harmful degree of cardiac excitement, but it saps the strength and lowers the tone of the nervous system. The medical attendant who has charge of a young man with valvular disease neglects his duty if he does not instruct his patient concerning the evils of sexual excess. I have known young married people of both sexes have their compensation seriously threatened after a few months of unbridled license in this regard. Although males are the chief sufferers in this respect, women with valve-lesions are often made to suffer through the inconsiderate demands of their husbands.

The tobacco habit has become so well-nigh universal, and youths are so often addicted to cigarette-smoking, that a few words regarding this habit are also indispensable. Young men who are just learning the seductive pleasures of tobacco should be strenuously urged not to form the habit, while those cardiac patients who are already addicted to smoking should be advised to discontinue it, or if unwilling to do that, to keep the use of tobacco well within the limits of harmful excess. Just how many mild cigars or pipefuls of mild tobacco a patient with compensated valvular disease may be safely allowed to smoke daily is a question impossible of general answer. The degree of indulgence permissible will vary in different cases, and must be determined by careful observation of the effect produced in each instance. The inhalation of tobacco smoke is most pernicious, particularly to individuals with mitral disease, since it will surely increase the already existing tendency to bronchial irritation. If it be true that smoking produces anæmia, then those persons whose compensation depends upon adequate nourishment of the heart-muscle cannot afford to have their red blood-cells impaired. It is well known that the immoderate use of tobacco occasions functional cardiac disorders, and, according to French authorities, raises arterial tension. Since, then, a healthy heart may suffer from tobacco intoxication, surely an unhealthy heart will experience the ill effects even more certainly and powerfully. When tobacco deranges digestion and causes insomnia, as it is well known to do in some individuals, its use should be peremptorily forbidden. I have heard it stated, with how much truth I know not, that Sir Morell Mackenzie was wont to say that the injurious effect of tobacco could be measured by its influence upon salivary secretion. In other words, when smoking causes salivation and frequent expectoration, it is an indication that the individual is too susceptible to its influence to safely persist in its use.

As regards the liquor habit, I do not propose to enter into a discussion concerning the food value of alcohol and its effect on the animal economy. Whatever be our views respecting the moderate use of liquor in its various forms, we all agree as to the evils of its excessive employment. What has been said already regarding the baneful effect of cardiac excitation in cases of compensated valve-lesions applies with added force to the immoderate consumption of alcohol. It goes without saying, that the possibility of destroying compensation is always present when a patient gives himself over to a debauch. I am a firm believer in the medicinal virtues of alcohol, but in the compensated stage of valvular disease the heart requires no medicinal treatment, and the only indication I can see for an alcoholic beverage is to promote the appetite and improve digestion of those individuals who habitually take too little nourishment for the requirements of their damaged hearts. The conclusion to which I have arrived, and which governs my actions respecting the matter under discussion, is that the young and vigorous with satisfactory compensation do not need alcohol in any form. Moreover, the danger of their becoming slaves to the habit is so real that the circumstances have to be very exceptional which make me incur the responsibility of recommending the use of even beer or wine to such patients. If one has been accustomed to a stimulant with his dinner then I do not interfere with his habit, contenting myself with a caution against its immoderate use. The habit of taking a hot toddy before retiring for the night is certainly not a good one, since, as pointed out by Fothergill, it accelerates cardiac action, and thereby robs the heart of some of its rest that ought to be obtained through the slowing of its contractions during sleep.

Marriage.—The baneful effects of one phase of married life have already been considered under the head of habits. There is another aspect of the subject which I propose to consider here. For the male who has been warned against and will avoid the dangers of a too ardent love, marriage is certainly advisable, since it conduces to regularity of living, and provides him with the comforts of a home that cannot be obtained in a boarding-house or hotel. In the case of a woman also it is better to be a happy wife with a comfortable home and a kind, considerate husband to minister to her needs than to be left alone, and possibly heart hungry. It is quite another thing if she is to become a domestic drudge, obliged to cook and wash for the family and to bear offspring. It is as regards the dangers of pregnancy and childbearing for women with valvular disease, even when compensated, that I wish to discuss marriage. Should a girl who has a valve-lesion become a wife? is the question often asked of the medical attendant. It is a most serious one to answer, and puts an enormous responsibility on the medical adviser. There are many cases of even compensated valvular defects in which pregnancy and childbirth are fraught with considerable risk. Yet every physician of experience can probably recall numerous instances of mothers who have successfully carried their offspring through to birth, in spite of serious heart-disease. Let us take up the valve-defects of the left heart separately.

Mitral disease, and of this mitral stenosis is the form most frequently met with in women. Theoretically, this is the lesion which should be the most seriously affected during the latter months of pregnancy and the expulsive stage of labour. As the gravid uterus rises in the abdominal cavity and when in the last two months its fundus interferes with the proper descent of the diaphragm, and crowds the viscera aside, dyspnœa and cyanosis appear, and walking often occasions serious distress. The right heart becomes embarrassed in consequence of mechanical interference with the circulation. The pregnant uterus impedes the descent of the diaphragm, so that the flow of blood out of the great veins and through the right heart into the lungs is deprived of the aid resulting from full and regular respiration. Moreover, the pressure upon the intra-abdominal veins retards the return flow from the inferior extremities, and blood-pressure in the capillaries is increased. This raises pulse-tension, and by thus increasing peripheral resistance throws greater work upon the left ventricle. Under the most favourable conditions this chamber discharges but a small volume of blood into the arterial system, and when its outflow is still further impeded by abnormal peripheral resistance, it results in augmented stasis within the left auricle, pulmonary system, and right heart. The vicious circle already existing by reason of the mitral stenosis and the strain upon the right ventricle are intensified. If this is not too severe, the woman may be able to endure her pregnancy to full term.

When at length labour comes on, and the expulsive stage is reached, there is imminent danger of the right ventricle giving way under the added stress of violent straining efforts.

The same condition obtains in mitral regurgitation, but the enlargement of the left ventricle, which if compensation is present is hypertrophied as well as dilated, is a factor for good by enabling the heart to withstand increased peripheral resistance. The augmentation of arterial tension would by raising intracardiac blood-pressure increase the regurgitation into the left auricle were it not counteracted by the left-ventricle hypertrophy. The danger is that the ventricle may not be able to resist the strain, in which event the evils of the mitral incompetence become intensified, and the right ventricle at length suffers from overstrain. Even if the injurious tendencies of pregnancy are successfully withstood, the woman with mitral regurgitation is subjected to the same danger during the second stage of labour as is one with stenosis. Whether the explanation just given is correct or not, the danger to mitral patients during this trying period lies in pulmonary engorgement and failure of the right ventricle. With this peril kept constantly in mind the attentive accoucheur can often conduct the pregnancy and gestation to a successful issue.

Capillary and venous stasis are to be lessened by saline or other not drastic cathartics and by keeping the patient at rest. When the expulsive efforts of labour endanger the integrity of the right ventricle or when stasis in the lungs leads to pulmonary œdema, instrumental delivery becomes imperatively indicated, and must not be delayed. In these cases chloroform is not attended with more than ordinary danger.

In cases of aortic-valve disease the strain of childbearing is on the left ventricle. Regurgitation through the aortic orifice is likely to be increased, and in aortic stenosis the augmented peripheral resistance hinders the output from the left ventricle in the same manner as if the orifice were for the time being still further contracted. Nevertheless, if compensatory hypertrophy is sufficient, the left ventricle alone, or chiefly, bears the brunt of the struggle. The patients as a matter of fact endure the ordeal of childbearing often without dangerous cardiac embarrassment and better than do mitral sufferers. According to Davis's statements, more than 50 per cent of mitral and 23 per cent of aortic cases succumb to the dangers of pregnancy and gestation.

Only yesterday I saw a woman in the seventh month of pregnancy who prior thereto presented well-marked evidence of mitral regurgitation with stenosis, considerable enlargement of the right ventricle, and dyspnœa of effort. Except upon walking for a considerable distance and in ascending stairs, this patient yesterday evinced no pronounced signs of cardiac embarrassment, and declared she was not specially inconvenienced by her pregnancy. Her pulse was only moderately accelerated, appreciably tense and strong, and the apex-beat was powerful, indicating adequate hypertrophy of the left ventricle. There was increased dulness to the right, but it was not excessive, and the right ventricle gave no sign of being dangerously overburdened. If this patient receives proper management during the remaining two months, and is not permitted to overstrain herself during labour, I believe her heart will not suffer damage from this, her eighth pregnancy. Her husband, who is a physician, states that she had heart-disease at the time of her marriage, fourteen years ago.

The following conclusions may be stated: (1) Pregnancy is a condition of gravity, but not necessarily of peril, to women with compensated valvular disease. (2) Labour is a time of real danger, the extent of which depends upon the nature of the lesion and the degree of compensation, but is often endured without catastrophe. (3) Mitral disease is more liable to disastrous consequences from both pregnancy and gestation than are aortic defects. (4) Even in mitral disease the degree of danger depends upon the state of compensation. (5) Pregnant women with valvular disease require special watching as labour approaches, and during the expulsive stage should be delivered instrumentally at the earliest indication of dangerous heart-strain. (6) The perils of marriage should be clearly stated to both of the contracting parties, and when compensation is imperfect or is maintained with difficulty they should be advised not to wed. (7) Interference with pregnancy is justifiable only when the nature and severity of the lesion render the maintenance of compensation impossible or when serious symptoms have already supervened.

Clothing.—The physician who would instruct his patient in matters of importance in maintaining compensation must have regard for what sometimes appear to be things of trifling moment. Among such details is to be included the clothing. All who have much experience in the management of cardiopathies come in time to realize the influence exerted by varying conditions of blood-pressure. The reason of one man's success, as contrasted with another's failure, in the treatment of heart-disease is often found in the close attention he pays to undue rise of blood-pressure. Take, for example, an ordinary case of mitral stenosis. Without any recognisable change in the cardiac condition or in his daily conduct, a patient will be conscious that his breathing becomes embarrassed by efforts usually put forth without any such effect. He consults his medical adviser, who, familiar with the case, discovers by studying the pulse and state of the venous circulation that there is unwonted tension in the arterial system. He finds, furthermore, that there is constipation perhaps, and knowing the influence of this condition over blood-pressure through the splanchnics, he administers a mercurial pill; tension within the abdomen and arterial system is lowered, and the patient's breathing returns to its usual state. A tendency to palpitation in a case of aortic regurgitation may be wholly due to increased capillary resistance and be relieved by the administration of a vaso-dilator.

It is because of the effect on blood-pressure produced by a patient's manner of dress that the matter of clothing becomes important, and may make for or against the establishment or preservation of compensation. Cardiac sufferers are often very sensitive to the cold and to sudden changes of weather, partly on account of a rheumatic diathesis and partly because of sluggish cutaneous circulation, as in mitral stenosis, or of relative arterial anæmia in aortic disease. Wool should therefore be worn next to the skin, and during seasons of low temperature the hands and feet must be kept warm by heavy not too tightly fitting gloves and shoes. It is well to have the latter constructed with cork soles, and in very cold weather overshoes may be a necessity. Outer garments should not only be warm, but they must not be so heavy as to tire the wearer. It is very important that the clothing does not constrict the vessels. This applies to shoes, gloves, collars, belts, and waistbands in the case of males, and to garters, corsets, and tight dresses on the part of females.. Constriction of the extremities tends to raise blood-pressure as well as to mechanically impede circulation, and should be corrected in every case of valvular disease. Harm is chiefly done, however, by garments that constrict the chest and abdomen. A woollen undervest that has become shrunken until uncomfortably tight, an overcoat that is outgrown, and can be buttoned only with difficulty, is in a measure at least injurious in the same way as is a too tight corset. Not only are respiratory movements hampered and venous circulation retarded in consequence, but the hypertrophied and therefore compensated heart is more or less compressed and restricted in its movments; abdominal viscera are engorged and displaced; the play of the diaphragm is limited, and the evil consequences of the valvular lesion itself are intensified. These effects are evinced by increase of cyanosis and shortness of breath, both of which disappear or lessen when the clothing has been removed.

Instead, therefore, of suspending the skirts from the hips, to effect which they have to be fastened snugly about the waist, it is preferable that women, particularly slender ones, wear garments of one piece, so that the weight may be borne by the shoulders; or they should replace the ordinary corset by a corset-waist, to which the skirts can be buttoned, thus avoiding constriction of the waist. Not many months ago I was consulted by a lady on account of attacks of dyspnœa and cyanosis, which at times amounted even to partial syncope. She presented signs of pronounced mitral stenosis with considerable secondary enlargement of the heart and hepatic congestion. She was inclined to corpulence, and to preserve her figure wore a long, stiff corset, which, in response to my inquiry, she declared was loose and comfortable. Not convinced on the point, I measured her waist both on the bare skin and outside of the corset, and thus demonstrated that when her corset was hooked she actually measured 4 inches less than she did next to the skin. I then explained at some length the harm she was doing herself, and succeeded in getting her to reform her mode of dressing, much to her relief, as she subsequently acknowledged. In this instance I am convinced that the symptoms of cardiac stress under conditions of exercise were due largely to the additional impediment to circulation and respiration occasioned by the tightness of her clothing.

Baths.—I have found in numerous instances that ladies with valvular disease were in the habit of taking a semiweekly hot bath, and some of them confessed to lying in the water for twenty minutes or more. Inquiry generally elicited the fact that the bath was followed by a feeling of languor, even amounting in some instances to prostration. It is well known that such hot baths are weakening to the heart and lower vascular tone. They should be forbidden therefore, and the patients advised to content themselves with a rapid sponge-bath daily if strength permits, and once a week a tub-bath of short duration, and of a temperature closely approximating that of the human body. Mitral patients endure bathing less well than do those with aortic lesions. Yet in all cases the degree of latitude permissible in the matter of baths is to be determined by their effect. If they are followed by a healthy reaction—that is, by warmth of the skin and a sense of rest or well-being—they are beneficial; but if a cardiac patient finds his bath leaves him with cold extremities and a feeling of fatigue, and that the exertion of briskly rubbing his body into a glow occasions breathlessness or palpitation, frequent bathing is likely to do him harm.

Swimming, whether in salt or fresh water, is to be considered not alone with reference to the temperature of the water; there is the shock of the sudden plunge or immersion, and also the exertion of propelling the body while at the same time sustaining the weight or pressure of the surrounding liquid. For these various reasons this form of bathing is apt to put a good deal of strain on the heart, and persons with mitral lesions, or those whose defects, of whatever nature, are maintaining compensation with difficulty, should either indulge in this sport not at all or only under great restriction, both of frequency and duration. It is probable that many of the cases of supposed death from cramp in the water are in reality instances of heart-failure or asystolism.

The Turkish and Russian baths and the various modifications of the shower-bath or douche found at health-resorts affect the heart and vascular system powerfully, and should not be taken by cardiac sufferers without the advice of a physician who is familiar with their effects and competent to decide on the advisability of such baths in each instance.

The saline and carbonated baths employed for therapeutic purposes at Bad Nauheim may be left out of consideration at this time, since they are not indicated for individuals whose valvular defects are in the stage of compensation.

Food.—Of all matters concerning our patients none is so essential as that of nourishment, and yet there is nothing, I venture to say, about which physicians of more than average intelligence and experience are so unable to give precise and suitable instructions. In works on diet are to be found tables of various foodstuffs compiled with regard to the needs of the human organism under conditions of work and repose, and from which one may construct dietaries suitable to the requirements of cardiac patients. In this chapter I shall only make certain general statements that apply to the regimen of persons whose lesions have not greatly deranged circulatory equipoise.

Adequate compensation in any given case implies, nay, necessitates, the supposition that the circulation of the digestive apparatus is not appreciably disturbed. The conclusion is warranted, therefore, that there is no digestive disorder secondary to the cardiac mischief and necessitating a corresponding modification of the diet. It is only essential that the food be of good quality, well-cooked, and sufficient. The proportion of proteids suitable to each case will be governed by the amount of exercise taken, the kind of work, and the tendency or not to obesity. The same considerations apply to the quantity as well as the quality. The proper preparation of the food is essential if digestive disorders are to be avoided. It is of importance also that meals be taken at regular hours; gluttony is injurious, and the amount of fluids taken with meals should be definitely stated, 10 ounces being ordinarily sufficient. Patients who are anæmic must be given a liberal allowance of beef, eggs, milk, and such vegetables and fruits as are rich in iron-forming compounds; those who incline to constipation are to get a dietary calculated to correct the tendency. Tea and coffee in moderation may be allowed. In a word, so long as compensation is complete there is no indication for special rules to govern the dietary further than what would be required for the preservation of health in the same individual were he not afflicted with an incurable malady.

Illnesses.—Among so many items of importance in the preservation of compensation it is difficult to specify one as greater than another, and perhaps it is not wise to attempt to do so. Yet I wish to lay particular emphasis on this point—namely, no illness or indisposition, apparently trivial in itself, should ever be so regarded in a person who has suffered injury from endocarditis. This is specially true of children. In them rheumatism is so apt to be masked that an infection of the throat, a persistent pain in an extremity, a rise of temperature without obvious cause, should always receive careful medical attention. The intestinal tract affords so ready and frequent a portal of infection that no deviation from the standard of health is to be neglected as of no consequence. Far better is it to bear the imputation of being over-careful and fussy than to some day awake to the consciousness that your neglect has permitted injury to visit one of your patients, whose compensated valvular lesion might otherwise have gone on years longer. Tell your patients emphatically and clearly that a tonsillitis, yes, or even an acute coryza, is never to be neglected. In a case of mitral disease, particularly of stenosis, an attack of simple bronchitis may break down utterly the integrity of the right ventricle. Be always suspicious of slight fevers, which to those living in a malarial region may appear to be of that nature; such a trivial yet persistent run of fever has only too often turned out to be an endocarditis. In cases of pneumonia and other serious affections cardiac sufferers merit more than ordinary care and watchfulness, if they are to come through undamaged. This is particularly true of persons with mitral defects not only because of the possible lighting up of a fresh endocarditis, but because the strain to which the right ventricle is subjected by reason of the valvular mischief is enormously augmented by the pneumonic consolidation—while at the same time cardiac endurance is impaired through the effect of the pneumonic toxines on the myocardium. If in such a case paralysis of the vaso-motor centres leads to cyanosis, the outlook is serious indeed. The gravity of pneumonia, la grippe, and other acute infections, gonorrhœa, diphtheria, scarlatina, measles, puerperal septicæmia, etc., in all cases of compensated valvular disease, is so obvious, particularly as respects the liability to fresh endocarditis, that further comment is unnecessary. Considerations of prophylaxis require, therefore, that our cardiopaths be properly instructed on these points. If it be objected that this is likely to render them introspective, and even hypochondriac, or if the patients are children, then the requisite information may be imparted to the family, friends, or parents. No one need fear that his motives will be misunderstood. Indeed, I am convinced that the American public likes to be talked to by physicians as if they were intelligent and reasonable beings, and that nothing conduces more to the establishment of confidence in the medical practitioner than frankness and plain dealing in all matters that concern the health of his patients.

Use of Drugs.—It is an error to suppose that every individual presenting signs of valvular mischief requires medicinal treatment. *Digitalis or some other heart-tonic is not to be ordered in every case in which an endocardial murmur is heard.* Inexperienced practitioners fresh from a medical college are very apt to commit this mistake, and consequently the foregoing injunction is not out of place. When a person with valvular disease presents himself in your office, inquire minutely into the matter of symptoms, and if he does not acknowledge any indicative of cardiac stress, remedies influencing the heart directly are not indicated. If you belong to that class who believe no person should be allowed to leave the physician's office without a prescription or a drug of some kind, lest forsooth the patient fancy he has not received his money's worth, then let it be a placebo. As a matter of fact, there are few persons who cannot be satisfied under such circumstances with an expression of opinion coupled with sound advice on the points under discussion in this chapter. Careful inquiry will usually bring out some pernicious habit, faulty digestion, constipation, some impairment of appetite, etc.; or there may be more or less anæmia, bronchial irritation perhaps, menorrhagia, some deviation from perfect health, which permitted to go on, will in time exert malign influence on compensation. Any such condition calls for correction, and to this end medicinal treatment may be indicated. What medicaments are suitable in each instance is, of course, to be left to the judgment of the medical adviser.

Although slight disturbances demanding attention are often dependent upon scarcely detectable disorders of circulation, they are not necessarily so in perfectly compensated cases, and hence these patients may generally be treated, so far as regards medicines, as they would be were they wholly free from endocardial defects.

Two conditions likely to prove more injurious to individuals with a valve-lesion, although compensated, than would be the case if his valves had not been damaged, are constipation and flatulent distention of the bowel. In both there is splanchnic irritation and consequent alteration of blood-pressure, but in the latter the effect of mechanical encroachment upon the contents of the thoracic cavity must be reckoned with. Uncorrected it may contribute materially to the destruction of heart adequacy, to say nothing about the patient's discomfort in the way of postprandial breathlessness in mitral and tendency to palpitation in aortic disease. Both disorders of digestive function tend to impair the appetite, give rise to neuralgias, anæmia, coldness of the extremities, and many other phenomena of auto-infection. Moreover, flatulent indigestion, probably through the absorption of toxines, is a frequent cause of deranged cardiac rhythm. This not only annoys or even alarms the patient, but it may even lead to the development of dilatation. It is for the relief of such disturbing factors as these, therefore, that drugs find their legitimate use in the management of compensated cardiopathies. In most cases the speediest and surest relief is likely to be afforded by a mercurial, a grain of calomel in divided doses, or 5 grains of blue mass, followed next morning by a Seidlitz powder or a glassful of some laxative water. Cascara, aloes, rhubarb, podophyllum, etc., pancreatin, ox-gall, nitrohydrochloric acid, subgallate of bismuth, salol or salophen, naphthol preparations, etc., alone or in various combinations and with which the reader is duly familiar, are serviceable, and will generally afford relief.

Only when gastro-intestinal disorders occasion persistent derangement of cardiac action are digitalis, strophanthus, caffeine, convallaria, and sparteine to be prescribed in this class of cases; even then the remedy is to be withdrawn so soon as the arrhythmia or acceleration of the pulse has been corrected. Temporary palpitation or vertigo on the part of aortic patients may generally be removed by minute doses of glonoin, $\frac{1}{200}$ to $\frac{1}{100}$ of a grain at intervals of two, three, or four hours, either alone or in conjunction with 3 to 5 drops of digitalis. When compensation has been seriously threatened by cardiac overstrain or some other disturbing factor, digitalis and strychnine may be needed in addition to rest and restricted diet; but in all such instances rest in bed for a few days is unquestionably the agency of greatest value, and should be rigidly enforced until the period of danger is past; not until then is the patient to be permitted to resume his usual mode of life and to return to his wonted exercise. In this class of cases "eternal vigilance is the price of safety."

Change of Climate, with Special Reference to High Altitude.—The question is so often asked whether a patient with heart-disease should go to Colorado or make the journey over the mountains to California, that it seems best to discuss this subject here when considering those conditions that make for the preservation of compensation. It is quite generally the opinion among medical men that the existence of a valvular lesion contra-indicates residence in elevated climates. This is too sweeping, as shown by clinical observation. My medical friends in Colorado assure me that their patients with valvular disease, of whatever kind, suffer no more inconvenience from their heart-lesions in Denver or Colorado Springs than do persons similarly affected at the sea level. Moreover, Regnard, in an elaborate work on the effect of high altitude on the heart and circulation, expresses the opinion that cardiac lesions *per se* do not contra-indicate residence in the mountains when this is necessary, and that aside from the discomfort of becoming accustomed to the high altitude individuals afflicted with heart-disease do not experience permanent harm. Nevertheless, he would not advise residence in such a climate for a cardiopath, since there is nothing in his disease calling for such a climatic treatment. This opinion impresses me as too broad, judging from the experience of some of my cardiac patients. I have known persons with mitral and aortic regurgitation to visit Colorado, and there, at an elevation of 6,000 feet, to take considerable exercise without discomfort, and apparently without harm. Others with vascular and cardiac degeneration have found the same to be true with them, and in fact one gentleman was actually able to walk with more ease at 7,000 feet than in Chicago. On the other hand, my patients who were not able to endure high altitude were sufferers from mitral stenosis, aortic stenosis, and mitral incompetence, when complicated by pericardial or pleuritic adhesions. These cases were all reported and discussed by me in a paper before the American Climatological Association in 1899, and will be found in the Transactions of that year. Singularly enough, Dr. Sewall, of Denver, in discussing my paper, stated that at that very time he had under observation in Denver a female with pronounced mitral stenosis, who had formerly been under my care in Chicago, and who was able to endure the elevation, not only of Denver, but also of Cripple Creek situated at an altitude of about 12,000 feet. She died a year subsequently, I understand, and I have often wondered if her residence at that elevation did not aid materially in shortening her life.

As the discussion of theories is foreign to the spirit of this work, I shall not discuss at length the considerations which make me conclude that residence in high altitudes is likely to prove injurious to persons having stenosis or regurgitant lesions complicated by pericardial or pleural adhesions. Suffice it to say that the effect of a rarefied atmosphere is acceleration and smallness of the pulse, together with increase in the depth and frequency of the respirations, the degree of this effect depending of course upon the degree of the altitude. The blood flows to the heart more rapidly, and if there is an obstruction at one of the orifices, this acts as a barrier to the rapid and easy passage of the blood, which tends to accumulate back of the hindrance. In mitral or aortic stenosis this would be in the lungs and right heart, and hence symptoms due to this engorgement are likely to result. If adhesions exist, they interfere more or less with the expansion of the thorax, which takes place in high climates, and consequently they ought to hinder that adjustment to altered conditions, which is essential if one is to become accustomed to a high altitude. Further reflection and observation since the publication of my paper on this subject have led me to the belief that probably all or nearly all individuals with valvular diseases can endure an altitude of 6,000 to 10,000 feet without injury, provided they do not take much exercise. Until they have grown accustomed to the rarefied air they should not walk at all, but remain in bed. At least such is the opinion and advice I give when consulted on this important question. Finally, it is probable that much of the dyspnœa and palpitation complained of by some at high altitudes is due in part, perhaps largely, to the fact that in the mountains the walks are not level. Hill-climbing is trying for cardiopaths, even at the sea level, and at an elevation where the air is thin such an exertion could readily prove doubly injurious. The phenomena of mountain-sickness—that is, rush of blood to the head, with vertigo, and even nausea, or in extreme cases bleeding from the nose and ears—are liable to attack healthy persons at too high an altitude, particularly during physical exertion. In their milder degrees they may affect cardiopaths in transit to and from the Pacific coast, but apart from these unpleasant symptoms patients who remain at rest in the car need not apprehend serious results even in passing the loftiest points. When vertigo or a tendency to syncope is experienced, relief usually follows the administration of a diffusible stimulant and the assumption of the recumbent position. Nevertheless, caution would suggest the taking of the less lofty routes.

CHAPTER XVII

THE TREATMENT OF VALVULAR HEART-DISEASE

(*Continued*)

II. COMPENSATION BEING IMPERFECT

A WANT of perfect compensation may have either one of two explanations. It may have never been adequately developed after the subsidence of the acute process that led to the valvular mischief, or having been once established it may be destroyed in consequence of a variety of causes. In the first instance the development of complete compensation may be impossible, owing to the extent of the damage sustained by the valves, in consequence of degeneration of the myocardium, or of the coexistence of complications or conditions residing in the patient's temperament, environment, etc. In such a case the course of the disease is generally too short to admit of its being brought into the category of chronic valvular affections. For the same reasons a long-continued maintenance of compensation may be impossible after it has once become established. In many cases, however, it is possible to arrest the downward tendency and restore heart-power when the injurious influences are discovered and removed. If these cannot be removed, then their baneful effects must be counteracted by all those therapeutic measures which are at our disposal.

In the management of compensated valvular disease the physician conducts a defensive campaign, so to speak, whereas when compensation has failed, he is called on to wage an active offensive warfare against all those forces that are striving to destroy his patient. His success depends not only upon the skill with which his therapeutic weapons are wielded, but also upon the precision of his orders, and the faithfulness with which these are executed. The treatment of valvular disease in this stage requires also attention to details of daily life, no matter how trivial they may appear to be, and the recognition of complications. Moreover, there are few patients in whose environment influences do not exist which, if permitted to go on, will not act unfavourably and retard the restoration of adequate compensation. For this reason these must be ascertained and removed so far as is possible. It is plain, therefore, that the proper management of these cases consists in much more than the mere administration of heart-tonics or other medicinal remedies. That highly gratifying results follow such strict and careful management is shown by the narration of the next three cases.

Miss X., referred by Dr. Minor, of Asheville, N. C., was first seen by me in July, 1898. She had spent the preceding winter in Asheville, and had there sought medical advice in the early part of summer because of increasing difficulty of breathing in walking, the altitude being 2,200 feet and the nature of the ground hilly. The winter immediately previous had also been passed in Asheville, but without her having noticed the same shortness of breath. She gave a history of inflammatory rheumatism three years before, and a year and a half before of an illness which, judging from her account, must have been an inflammation of a serous membrane within the thorax. Her age was twenty-two. Her pulse was much accelerated, 120 or more, regular, and equal. The ankles were œdematous, and the abdomen was distended by the greatly engorged liver. The broad, fairly powerful apex-beat was found immovably fixed in the sixth intercostal space, near the anterior axillary line, and superficial cardiac dulness extended somewhat beyond the right sternal margin, upward to the third costal cartilage and at the left, almost to the mamillary line. There was an intense blowing systolic apex-murmur transmitted to the left and accompanying, not replacing, the first sound, the pulmonic second sound being very accentuated. Upon auscultation, fine crackling râles were detected during inspiration along the upper and left border of præcordial dulness, and upon the arms being raised and lowered a creaking sound could be plainly heard at the superior boundary of dulness on the sternum.

In this case it was manifestly not so much the mitral leak that was serious, as it was the fixation of the left ventricle that was preventing the maintenance of compensation. Moreover, it was foreseen that digitalis and similar remedies could exercise but limited control over the heart, since only slight if any reduction in the dilatation of the left ventricle was possible by reason of the restraining adhesions. Efforts had to be directed, therefore, to lessening the resistance residing in the congested portal system and at strengthening the right ventricle. The former was to be accomplished by purgatives, tonic doses of digitalis, and the removal of all constricting clothing; the latter by those same means, re-enforced by abstaining from too much physical exertion, and if need be by rest from all exercise. Accordingly the patient was told to give up her corset, which she did, to take an aperient water daily before breakfast, and thrice daily 15 drops of tincture of digitalis. Stair-climbing was forbidden. Directions regarding the quantity and kind of food and the amount of fluids were added. The degree of improvement was so insignificant during the next few weeks that at length absolute rest in bed was advised and acted upon. Then benefit became at once apparent in slowing of the pulse and diminution of dulness over the right heart. The liver also began to shrink, urine grew more abundant, and œdema disappeared. After five weeks of enforced rest the patient was permitted to gradually resume her ordinary mode of life, excepting that she was not allowed to go out. All this time digitalis or strophanthus was continued in about the same dose (15 drops) of the one and 10 drops of the latter thrice daily, with exception of every sixth day, when it was omitted. Apex-impulse grew somewhat stronger, but never altered its position in the least; the marked change was in the right ventricle and liver.

After a few weeks longer of such management it was decided to try the effect of a course of Nauheim baths. They were given in her home and not at my rooms, as was then the rule with patients whom I subjected to this treatment. Whether because I could not watch their effect as closely in this way, or on account of the hampering effect of the adhesions (I believe it was the latter), the baths did not produce a beneficial effect. Heart-rate increased and showed a tendency to unsteadiness, dulness to the right became increased, and the amount of urine diminished. They were discontinued, therefore, and no permanent harm resulted. Considerable difficulty was experienced in getting this patient to give up her candy and obey instructions as to diet; but the habit of making her furnish me with an account of what she ate and drank finally convinced her that I was in earnest, and now her obedience to orders is all that can be desired. In this case menstruation is too profuse and somewhat irregular; so that she has been instructed to remain in bed during her menses, and hydrastin hydrochlorate is sometimes administered. During the summer of 1899 her health was quite satisfactory, and she was able to enjoy a number of outings with friends without very irksome restriction on her pleasures. She has been very subject to annoying pains in her shoulders, but especially in her chest beneath the sternum, and upon several such occasions there has been a circumscribed, faint, yet distinct friction directly above the superior line of cardiac dulness, which was taken to indicate a fresh lighting up of mediastinitis. These attacks have generally yielded to counter-irritation and antirheumatic remedies.

Towards spring of 1900 her condition began to run down slowly but surely, and complaint of "rheumatic pains" was frequent; the pulse quickened, and her flesh grew flabby and cold. She consented to enter a hospital, where her temperature could be carefully watched, as the possibility of endocarditis was entertained. Temperature remained subnormal, however, rather than febrile. Her urine was collected and examined, with the following result: Total amount in twenty-four hours, 660 cubic centimetres; specific gravity, 1.028; urea, 3 per cent; 2¾ per cent of albumin; numerous hyaline and granular casts. Animal food was withheld, copious draughts of water insisted upon, and citrate of potash was administered, together with moderate doses of foxglove in tincture. A week later the urine amounted to 2,500 cubic centimetres, had a specific gravity of 1.013, urea 1.3 per cent, neither albumin nor casts. This must have been a mild nephritis and not merely congestion, since the kidneys responded so well to the free intake of fluids. As was to be expected, the patient's condition improved rapidly, the pulse-rate decreased strikingly; and two weeks afterward she returned home. It is now four months later, and albumin has not again been found in the urine. She has been allowed animal food but sparingly, and has been required to drink water freely between meals, with the result of her feeling unusually well, and being able to enjoy a moderate amount of walking again. This lady is an invalid, to be sure, who has to lead a restricted existence; but she is able to attend matinées and social functions of a quiet kind, in fine, to get a good deal of enjoyment out of life. She cannot entertain the hope of becoming a wife and mother, and has been so informed.

In this case digitalis has been taken most of the time, because it has been repeatedly proved by trial that when discarded entirely its need is shown after a few days by increased breathlessness and subjective as well as objective rapidity of the heart's action, to 120 or more. The tincture has generally been prescribed, sometimes 10 drops once daily, and at other times 5, 7, or 10 drops two or three times a day. Upon a few occasions scantiness of the urine and œdema, shown by pitting of the ankles, has necessitated the administration of the fresh infusion of English leaves, a tablespoonful three or four times daily. Now and then citrate of potassium has been added. Sulphate of strychnine has also been taken much of the time, and whenever she has felt more than usual weakness she has profited much from the compound syrup of hypophosphites.

She has been dependent on medicinal remedies, but no doubt a large part of her really good condition is owing to the excellent care she has taken of herself. She has dressed sensibly, wearing a loosely fitting corset-waist, and so far as possible suspending her skirts from her shoulders. Walking has been done at a slow pace and for short distances, care being taken not to walk against a strong wind, and to stop for rest whenever shortness of breath or palpitation has been experienced. Ascending stairs has been avoided, or when that was impossible they have been mounted a few stairs at a time, with frequent pauses to let her heart quiet down or to recover breath. During stormy weather she has either remained indoors or has driven in a closed carriage to her destination. Any indisposition, however trivial, has received prompt attention, and a day of unwonted fatigue or exertion has been generally followed by rest in bed or on a couch. Her dietary has been simple and nutritious, and she has not been permitted to be in the least constipated. On the contrary, she took an aperient water every morning for a year at least, with a dose of calomel whenever her liver showed more than ordinary congestion or she felt a sense of fulness about the waist. Latterly the laxative water has been taken only every other day. Finally, she has been required to see me at regular intervals, generally two or three times a month, that thereby she might be kept under control. In her case certainly eternal vigilance has been the price of safety.

Master W. B., aged eight, was examined by me in June, 1896, at request of Dr. John Streeter. He was a frail, undersized, pale boy, whose whole life had been one of many illnesses; bronchopneumonia five times in early childhood, innumerable attacks of fever, with coated tongue, pain in the right hypochondrium, nausea, and irritable stomach, which had generally yielded to calomel and milk diet, and had been considered "storms of uric acid." For a year prior to my visit patient had been under treatment by Dr. M. Allen Starr, of New York, who, it was stated, had administered bromide of soda every night during the year. The mother had first learned of her boy's heart-disease in April, 1896. Patient was very subject to attacks of acute tonsillitis, having but just recovered from one, and at the time of my examination had an acute coryza. He had the facial appearance indicative of adenoids, breathed through the mouth, and beneath the angle of the left inferior maxilla the neck was tumefied by enlarged cervical glands. His chest was long, narrow, sunken below the clavicles, prominent in the præcordium, and expanded poorly on inspiration; the finger-ends were noticeably bulbous, but there was no cyanosis. The heart was greatly enlarged, there was a loud, harsh systolic apex-murmur of wide propagation, and both liver and spleen were palpable with much corroborative increase of dulness. The little fellow suffered from dyspnœa and occasionally palpitation on ascending stairs or hurried walking. Urine was scanty and of high specific gravity, otherwise negative. His appetite was capricious, digestion weak, and bowel movements irregular.

It was my opinion that, if much improvement was to be attained, three things would have to be done—the removal of the nasal obstruction, the development of the chest, and the improvement of the blood condition. The adenoids could have been safely removed under ether and even chloroform skilfully administered, and thus the first step taken towards proper expansion of the chest. I have notes of a similar condition in a boy of five or six, which was successfully operated on without the slightest untoward effects as regarded his mitral insufficiency and with ultimate benefit to the child. In the present case the mother wanted the treatment of the nasal obstruction postponed, and I have never learned whether it has been done or not. It was evident that if the already enlarged heart was to have room in the thorax for further increase of its compensatory hypertrophy the capacity of the chest would have to be augmented. Accordingly, his attendant, an intelligent trained nurse, was instructed how to give resistance gymnastics and breathing exercises. These were intended not alone to strengthen his heart and develop his thorax, but also to facilitate blood-flow by better aspiration up out of the congested liver and abdominal vessels. A highly gratifying experience in other cases had already shown how effective and beneficial such exercises are in such a condition.

In addition, improved nutrition was sought to be achieved through a dietary suited to his blood-state and to his feeble digestive and assimilative processes. Starches and sugars were greatly though not entirely cut off, such as were allowed being carefully selected—zwieback, toast, a little baked potato, etc. Meat and eggs were allowed in moderate amounts, and certain fresh vegetables and fruits were added to the diet list. Such medicinal remedies as would aid digestion, keep down fermentation, and unload the portal vessels were prescribed. To the last end calomel was the drug selected, care being taken not to produce a too powerful purgative effect. Cardiac tonics, digitalis and strychnine, were a minor part of the treatment, being administered in such doses only as would gradually tone up the heart. Some improvement began to be apparent almost directly, but the family removed to the East before time was afforded to observe the ultimate results. Information came to me, however, some weeks subsequently that the plan of management detailed was bringing about improvement. I have not seen the patient since that time.

W. H. W., aged thirty-nine years, male, physician, consulted me in August, 1896, on account of an attack of mild articular rheumatism, one week previously, in right knee and both hips. He gave a history of inflammatory rheumatism at age of nine or ten, at fifteen remembers he had shortness of breath, and thinks he had intermittence. In 1880 valvular disease was diagnosticated. During 1895 he had an afternoon temperature from 99° to 100° F., but the cause was not discovered. In December, 1895, had a fever of 103° F. that lasted three days, and yielded to rest in bed and milk diet. Afterward felt better than before. His condition was good the following winter, and until his recent inflammatory attack he has attended quite constantly to an exacting general practice. At the date of my examination there was slight dizziness on walking, temperature at 3 P. M. was 99.8° F., pulse was 98, sitting, falling to 94 on assuming the dorsal decubitus, and was collapsing; capillary pulse was present, and there was a systolic snap in the femoral artery. The broad, strong apex-beat was in the sixth left interspace, 3½ inches to left of sternum, and there was a diffused systolic impulse over the body of heart to left of the breastbone. First sound at apex was prolonged and impure, suggesting a presystolic murmur, while the aortic second was muffled. In the aortic area was a soft, faint diastolic murmur, transmitted downward and to the left. The diagnosis was plainly an aortic insufficiency of rheumatic origin, and a still persisting mild rheumatism.

He was advised to give up active exercise so long as any trace of joint inflammation persisted, and to take salicylate of soda with small not frequent doses of digitalis. The patient subsequently reported his recovery and return to practice. During the ensuing three years he abandoned general practice and limited himself to office work; he moved his residence a short distance out of the city, which necessitated travelling to and fro on an elevated road, and the ascending of long, steep stairs to the stations. He consulted me several times, and on one occasion reported an attack of hæmoptysis following some exertion. After that attack I confined him to bed for a week or so, and put him on digitalis, strychnine, and a vaso-dilator. He quite frequently experienced intermittence of the pulse for days together, and as he had some digestive disturbance at those times, it was thought the intermissions were due to that cause. On one occasion, in the latter part of 1898 or the beginning of 1899, he was suddenly seized with partial syncope while at work over a patient in his office, which, however, was recovered from after a few days rest at home, with the use of digitalis and nitroglycerin.

At length, in January, 1900, he entered my office one morning saying he had just had a quite profuse hæmoptysis without the provocation of unusual physical effort. The heart was hurried and occasionally intermittent; its left border was much outside of the left nipple, its apex rather too rounded, and its impulse not well defined. It was also noted that a rough systolic murmur had developed in the aortic area, which had not existed a year or two previously. The condition was considered very threatening, as the hæmoptysis pointed to pulmonary congestion in consequence of temporary inadequacy of the left ventricle. The emphatic admonition was given him to return home at once and go to bed for an indefinite time, probably many months. The advice was acted on, and he began the regular employment of small, thrice daily, doses of tincture of digitalis, with 3 grains of potassium iodide t. i. d., and strychnine; glonoin was substituted now and then for the iodide.

His dietary was light yet nutritious, and the bowels were kept free by calomel and other laxatives, as occasion required. For a short period he had a light run of fever, with vague joint pains, which yielded to salicylates. His cardiac action was invariably irregular after breakfast, but subsequently grew less annoying or disappeared entirely after his morning glass of milk was abandoned, and his early meal was made more substantial. This patient remained in bed for four months, at the end of which time his heart was found to have retracted somewhat in size, gained in the force and concentration of its apex-beat, and had become noticeably steadier in action. He was then permitted to resume exercise very gradually, at first about his room, and thus by slow degrees to accustom himself to his ordinary habits of life. When at length he had grown able to get about as before his illness, he went into the country, and there, driving about with a medical friend, soon got to feeling as well as usual. Small doses of digitalis and strychnine were continued after he left his bed and resumed walking, for the purpose of maintaining what the heart had gained by the prolonged rest. Considering the age of this patient (now forty-three) and his history of repeated subacute rheumatism and probable aggravation of the endocarditic changes, the results secured were highly gratifying, and illustrate the immense value of physical inaction in the recumbent posture in cases of aortic regurgitation with breaking compensation. This patient has had no return of his symptoms, so far as I have learned, up to the present writing. Nevertheless, the prognosis is not encouraging, for unless the doctor is very careful a final and irretrievable breakdown is likely to occur at any time.

Medicinal Agents.—From the narration of the foregoing cases it becomes apparent that the principle of management applicable to the stage of compensation does not obtain when heart-power shows signs of failure. In this stage digitalis or one of its congeners is generally of great service, and is often indispensable for the remainder of the patient's life. Foxglove is incomparably superior to all cardiac tonics of its class, and should always be preferred so long as vascular changes are not present and when it does not disagree with the stomach. The former objection does not exist in the young and in some persons at or past middle age. When the arteries are stiff and the vaso-constrictor effect of digitalis is likely to occasion injurious rise of blood-pressure, this effect can be overcome by the administration of $\frac{1}{100}$ of nitroglycerin every two or three hours in the form of a tablet of required strength or a minim of the official solution. Two or three grains of an iodide salt are said to accomplish the same purpose, and may be administered three times a day. If strophanthus is employed instead of digitalis, a vaso-dilator may or may not be necessary, according to the degree of vascular tension. The unpleasant effect of digitalis on the stomach is said to reside in a free-fat and certain narcotic principles, the irritating qualities of the drug in free acids, all of which can be removed without impairing its efficiency. The method of removing these objectionable constituents was announced in 1899 by Dr. England, the chemist of the Philadelphia Hospital. Accordingly, such a fat-free tincture of digitalis is now prepared by several manufacturers of pharmaceutical preparations, which has been found to possess equal if not greater potency than the tinctures ordinarily in use. In most cases of the kind now under consideration digitalis is needed for its tonic effect, not as a diuretic, and therefore the dose may be a moderate one—5, 10, or 15 drops of the tincture once, twice, or thrice daily, as the case may be. The length of time during which digitalis is to be administered is also variable. Usually, however, it will be required for many weeks or even months; in grave cases it may even be continued for the rest of the patient's life. I am convinced that a digitalis-habit may be acquired, yet see no objection to this so long as the continued use of the remedy prevents a total loss of compensation.

Another medicinal agent of generally recognised value as a cardiac tonic is strychnine. It stimulates the heart through its action on the cardiac motor ganglia. The slight retardation of the pulse-rate, which is produced by its stimulation of the inhibitory apparatus, is transient, and therefore not to be reckoned with in considering its therapeutic influence. The increase of arterial tension, said to result from its stimulation of the vaso-motor centres, is so slight that opinions are at variance on this point. This effect is certainly too trifling to prove an objection to its employment, even in cases showing pronounced vascular degeneration and consequent high and sustained pulse-tension. The question of prime importance is, In what dose is strychnine to be administered? Believing that if it stimulates cardiac contractions in small doses through its action on the motor ganglia, it ought to do this still more powerfully in large ones, I have been in the habit of ordering doses that to many seem dangerous—that is, I have many times prescribed $\frac{1}{30}$ of a grain hypodermically every three, and even every two, hours, until seven or even eight injections have been given in a day, and have continued these doses for days, and even weeks together without ill effects, so far as I could discover. On the contrary, they have seemed to be of positive benefit. Indeed, I may say it never occurred to me that the remedy, even in these doses, could do more harm than occasion the primary phenomena of its physiological effect. As I have but rarely observed twitchings to result, and in these cases have promptly discontinued the drug, I have not thought to question its beneficial action. In a recent conversation with Dr. R. G. Curtin, of Philadelphia, I was surprised to find that he strenuously objects to such large doses on the ground that it is likely to produce short and irritable systoles instead of long and strong contractions of the ventricle, such as are required to drive the blood onward energetically. He thinks that the neurility of the cardiac nerves and ganglia become exhausted. He stated, moreover, that he was gratified to find, during a recent visit abroad, that such experienced clinicians as Ernest Sansom and Lauder-Brunton do not exhibit the agent in large doses, contenting themselves in fact with $\frac{1}{60}$ of a grain three or four times daily. Such opinions are worthy of consideration, and are here given in the hope of stimulating original observation on this point. It is difficult to abandon notions that have dominated one for many years and seem to have the support of favourable experience. I feel sure that under the influence of such large and frequently repeated doses I have seen a weak heart rally and evince signs of augmented power. I have certainly known a dying heart to be kept beating for hours and days by the combined use of strychnine and nitroglycerin after speedy death seemed inevitable. There can be no doubt of patients becoming so dependent upon this medicine, when taken for a long period, that they develop a strychnine habit, the same as a morphine habit. Only the former is not so harmful nor so difficult of abandonment.

Whatever may be the answer to this question of large or small dosage in cases of dire urgency, I would not wish to be thought to advise them when cardiac power is only beginning to fail or cannot be said to be entirely competent. In the stage now considered it would probably suffice to prescribe $\frac{1}{60}$ or at most $\frac{1}{40}$ thrice daily. The length of time during which this agent is to be continued must depend upon the circumstances of each case, and therefore is to be left to the judgment of the medical attendant.

The value of the nitrite compounds has already been stated in speaking of the vaso-constrictor effect of digitalis. It may be said in addition that these agents are often highly beneficial in the treatment of aortic regurgitation even when digitalis is not indicated. The earliest premonition of failing heart-power in these cases is sometimes shown by attacks of vertigo, and occasionally by syncope, in other instances by a "pounding action of the heart," to quote the language of the patients. These symptoms are an indication that arterial tension is outstripping the contracting force of the left ventricle, which is consequently unable to successfully cope with the heightened peripheral resistance. Digitalis augments the vigour of cardiac systole, but it also still further raises arterial tension, and hence may increase rather than lessen the tendency to palpitation. It is better, therefore, to try the effect of $\frac{1}{100}$, or it may be less of nitroglycerin three to four times daily for the removal or reduction of undue vascular tension, in the hope that the symptoms will disappear without recourse to digitalis or strophanthus. Glonoin stimulates the heart only indirectly by causing vaso-dilatation, and thus removing obstacles in its path, so to speak. Excepting, therefore, as a vaso-dilator, nitroglycerin is rarely to be employed in this stage of valvular affections.

A perusal of the cases narrated in this chapter will impress the reader with the great benefit often derived from cathartic remedies, and the important rôle played by them in the management of patients. Their utility was first really impressed upon me by the writings of English authors, and to their teachings I owe much of my success in the management of cardiopathies. I shall have more to say on this subject farther on. It will suffice at this time to direct attention to the tendency of most valvular lesions, especially mitral and those of the right heart, to congestion of the veins of the abdominal viscera even before signs of compensatory disturbance grow pronounced. These congestions cannot be so surely and quickly relieved by any other means; often they cannot be removed at all without recourse to purgatives.

If all that was needed was to increase the driving force of the left ventricle, and thus to push the venous blood onward, then digitalis would be the remedy *par excellence.* In these valvular diseases, however, there is an impediment to the flow of venous—i. e., of the return blood through the lungs and heart. Behind this impediment the circulation becomes dammed up. The surest mode of preventing an inundation is to provide an outlet, and this is done by carrying off some of the water of the blood through the intestines. When this has once been accomplished, then a heart-tonic or stimulant may be able to reinstate a satisfactory degree of circulatory equilibrium. In some cases it is impossible to do more, or even hope to do more, than keep the stasis within bounds and render the heart's labour somewhat easier. Aloes, cascara, etc., which unload the colon relieve constipation when it exists, but they do not occasion free watery stools, such as are needed to deplete the engorged portal and tributary veins. To this end, saline preparations or such other drugs as are not too drastic are required. Of these, nothing is more efficient than sulphate of magnesia in saturated solution or dissolved in hot water and taken half an hour before breakfast. Its taste is very objectionable to some persons, and it is sometimes rejected by a sensitive stomach. In such an event it is better tolerated if to it are added half a dozen minims of the ordinary essence of ginger kept in every household. Four ounces of the compound infusion of senna, the familiar "black draught" of the English, make a very potent and not especially disagreeable hydragogue cathartic. Pulvis jalapi compositus is also highly efficient, and by me greatly esteemed. A teaspoonful may be taken by the average individual, whose venous stasis is pronounced, without his being unduly weakened thereby. There are many other remedies having a similar action, of which space forbids mention.

Of all, however, there is nothing which will ordinarily produce such happy results as calomel or blue pill. That they powerfully affect the circulation and promote excretion is shown by the diuresis they promote even before they have emptied the bowel. It is generally well to administer the mercurial at bedtime, and have it followed next morning by a saline. The frequency with which such cathartic medication is to be employed will have to be determined by the degree of stasis and the diminution that ensues.

Patients with valvular disease are often anæmic, either because the liver is unable to utilize nucleo-albumins in the manufacture of iron, or in consequence of the destruction of hæmoglobin by some ferment generated in the intestines. The so-called hæmatics, iron, arsenic, and the hypophosphites, would appear to be indicated, therefore, and certainly do act as a tonic, but to my mind it is doubtful whether their beneficial effect is not due to their improving appetite and digestion rather than to their directly increasing the percentage of hæmoglobin.

Medicines that always appear to me to be of positive utility are all those that facilitate the better digestion of food and lessen the likelihood of gastro-intestinal fermentation. These are pepsin, pancreatin, taka-diastase, dilute hydrochloric acid, the simple bitters, and the various antiseptic remedies, salol, salophen, benzonaphthol, etc. The use of these agents, together with the improved function of the digestive organs incident to the relief of stasis by catharsis, has always seemed to me to do more towards the lessening of the spanæmia than do iron and arsenic.

Rest.— Leaving now the consideration of medicinal remedies, we come to certain other factors that are of utmost importance in the restoration of compensation, and of these rest takes the first place. It is universally recognised by practitioners that for weakened hearts no measure is so beneficial as physical repose in the recumbent position. Not so with the laity, and patients frequently persevere with some form of exercise in the mistaken notion that thereby they will regain strength. So soon as a heart that is damaged by endocardial disease exhibits signs of being sorely overtaxed, physical exertion should be interdicted and the patient put at entire rest until conditions are improved. The reasons for this are not far to seek, being found in the mechanical effect on the circulation and in the resulting improvement to cardiac nutrition.

When in valvular disease compensation is imperfect, absolute physical rest for several weeks seldom fails to prove highly beneficial. The heart is not compensating, because it is being overtaxed by having to receive and discharge more blood than it can handle easily. If in such a case the patient is put to rest, active muscular movements are abolished and respiration is less rapid and more shallow. Venous blood is delivered to the right auricle less rapidly and the right ventricle is given less work to do. Cardiac contractions become less frequent, but more efficient, and its chambers are better able to empty themselves. Thus the decreased inflow and the increased outflow tend to diminish dilatation and promote the re-establishment of that preponderating hypertrophy essential to compensation. Improvement of circulation is shown by the better quality and rhythm of the pulse, by the reduction of signs of stasis, and by augmented excretion of urine. There is improved visceral function in general, and there is better nutrition of the whole body as well as of the heart-muscle. This latter, which is of great importance if cardiac power is to be maintained, also results directly from the fact that physical repose favours a better coronary circulation.

With the slower action induced by physical inactivity the heart tends to gain in power, and the left ventricle to discharge a greater blood-wave into the aorta. The coronary arteries are better filled, and the heart-muscle receives a supply of blood more adequate to its needs. This, however, is but a part of the benefit to the heart proceeding from enforced rest, particularly in cases of mitral and aortic obstruction. It has been explained how this treatment lessens cardiac dilatation. It is the right heart chiefly that profits in this way, the ventricle emptying its contents more completely, and stasis in the auricle being diminished. This now acts favourably on the circulation in the coronary veins. With lessened intra-auricular blood-pressure resistance to the outflow from them is less, owing to the fact that they empty into this auricle. Stasis within them tends to subside, and with a better circulation the products of cardiac metabolism are more fully removed.

Let us now consider the benefit resulting from rest in the individual valve-lesions of the left heart. In mitral stenosis there is practically a dam built across the blood-stream at the point where the blood coming from the lungs is poured into the left ventricle. So long as compensation exists the hypertrophied left auricle and right ventricle are able to discharge over this pathological dam—that is, through the narrowed mitral opening—so large a portion of the blood sent through the lungs that serious congestion within the pulmonary vessels does not take place. When compensation begins to fail, and cardiac contractions to grow more rapid, the diastolic pause, during which the left ventricle is expected to fill, is shortened, and time is not allowed for the left auricle to empty its contents.

Stasis begins in the parts back of the stenosis, and grows ever greater with the progressing loss of compensation. Something must be done to diminish the rapidity and volume of the stream pouring into the left auricle. This is precisely what is accomplished by rest. Diastoles are lengthened, more time is given for the filling of the left ventricle, which consequently throws a larger quantity of blood into the arterial system, and there is a tendency to restoration of the proper balance between the aortic and pulmonic systems, on the one hand, and the great arterial and venous systems on the other.

In mitral incompetence there is a systolic reflux into the left auricle, and the stream entering this chamber from the lungs is momentarily checked, to be the next instant unimpeded as diastole succeeds systole and the blood gushes into the ventricle. Yet, while there is a momentary checking of the flow in the pulmonic vessels and an inevitable tendency to back-pressure, the column of blood into the left auricle and pulmonary veins, together with the walls of these vessels and of the auricle, serves to resist the regurgitant rush from the ventricle. So long, therefore, as this resistance is effectual cardiac adequacy is unimpaired, and evidences of stasis are wanting.

When this compensation begins to fail, it is necessary to relieve the walls of the left auricle, the pulmonary vessels, and the right ventricle from overstrain by lessening the frequency of regurgitation and by retarding the flow from the systemic veins into the heart and lungs. Rest accomplishes this, and thus proves a powerful factor in the resumption of heart-power and the removal of stasis.

In the same way also as in mitral stenosis the coronary veins are better emptied and the coronary arteries are better flushed, nutrition of the heart-muscle is improved, the aortic system receives more blood with each systole, and an improved general nutrition results.

When in aortic obstruction compensatory hypertrophy of the left ventricle begins to yield to dilatation, the contents of the ventricle are no longer adequately driven through the stenosed orifice. Signs of stasis appear and increase in proportion to loss of compensation.

Two things are now required if the threatening breakdown is to be averted: (1) More forcible contractions on the part of the left ventricle, and (2) the delivery of less blood to the ventricle. Rest slows the heart by lengthening its diastoles, and but little if at all its systoles; while if it affects the vigour of the latter, it does so only indirectly by relieving it of strain and improving its nutrition. It can do very little, therefore, towards enabling the left ventricle to drive blood through the narrowed aortic orifice, and, moreover, experience has taught that when in this disease the left ventricle begins to weaken, it is an indication that the stenosis has overpowered the ventricle. All that is left is to spare this chamber as far as possible. It is by accomplishing this, or the second requirement mentioned above, that rest is of service in aortic stenosis. It serves to retard the flow of blood into the left ventricle, and thus to lessen the amount which this chamber is required to discharge past the point of constriction. Therefore, although this therapeutic measure is of service in conserving heart-power in this affection, it cannot accomplish such brilliant results as in mitral disease.

In aortic regurgitation failing compensation means impaired resistance on the part of the left ventricle to the distending force of the regurgitant stream, a still more imperfectly sustained blood-pressure in the arterial system, and after a time secondary overfilling of the veins, right heart, and lungs. The danger lies in sudden diastolic arrest of the left ventricle while the mitral valve is still competent, or in such a dilatation of the ventricle that relative mitral insufficiency with all its consecutive evils is produced. The yielding left ventricle must therefore be relieved of dangerous overstrain. Inasmuch as physical exertion and the erect position are thought to raise intra-aortic blood-pressure and intensify the regurgitation, the removal of these injurious, even dangerous, influences becomes imperative. This can only be accomplished by a rigid, and often prolonged, confinement in the recumbent position.

There are also two other reasons for insisting upon rest in these cases: (1) Physical inaction slows the flow of blood in the systemic veins, and thus tends to check the discharge into the ventricle from the left auricle. With this stream, as well as the regurgitant one reduced, the disabled ventricle is called on to handle less blood and finds its labours diminished. (2) Rest of body means also rest to the heart, since by slowing down its contractions its diastole or period of repose is lengthened, while the actual amount of work required of it is reduced.

It may be argued that the lengthening of diastole favours a better filling of the ventricle, and therefore compels it to put forth greater effort in order to discharge this larger amount of blood. This would be so if the flow to the left auricle were not retarded; but this latter being the case, there is not so much likelihood of overfilling the ventricle as when the patient is up and active. This consideration, however, renders it probable that the chief benefit of rest lies in the rest to the heart-walls and in the less forcible reflux from the aorta.

The mechanical conditions existing in this lesion, and the nature of the pathological changes that take place in the myocardium, render prognosis exceedingly grave whenever a case of aortic incompetence shows signs of failing compensation. The probability of restoring heart-power is so slight that any means, however unpromising, should be made the most of. Accordingly, rest of body and mind must be enforced with greatest rigour and for an indefinite length of time, not merely for weeks, but for many months. As a matter of fact, the prospect of regaining cardiac power in serious loss of compensation is poor, and rest is of service mainly in prolonging life.

Since, then, rest is so valuable a means of treatment in our attempt to preserve or restore cardiac adequacy in uncompensated valvular disease, the physician must not content himself with partial obedience. If the case is urgent, he must see that his orders are carried out faithfully. When a patient is told that rest in bed is needed, he must be made to understand that by it is meant not rest for a few hours each day, but rest both day and night. Moreover, it does not mean that he can get up as often as he pleases to fetch some article he wants or to walk to the toilet, that is situated perhaps a short distance down the hall. It means that he is to remain in bed, and is to have the attention of a nurse who can spare him all avoidable effort.

Patients suffering with aortic insufficiency require more rigid enforcement of absolute rest than do most persons with mitral disease. A single indiscreet effort may undo all that has been gained by weeks of inaction. Therefore, such a patient who is struggling to preserve his left ventricle from complete destruction must lie as quiet as possible, making use of the bed-pan and urinal bottle, and taking his meals in the dorsal decubitus. If this is impossible, as is sometimes the case with nervous individuals, then they may be lifted a little higher by the nurse, and, supported by pillows, may take their meals in this position. Better yet is the adjustable bed, which permits every possible position, without the slightest exertion on the part of the patient.

Of course each case has to be treated on its own merits and according to its own exigencies. One patient may be permitted partial rest, and yet do well, while another may require the strictest enforcement of this principle of management. In some cases, also, the attempt to carry out rigid confinement to bed for months, no matter how important it may be, is sure to create such a spirit of restlessness and discontent as will counteract all that is gained by physical repose. It is evident, therefore, that judgment and tact are often required in the enforcement of this therapeutic agency.

Finally, when asked, as he is sure to be, how long rest is necessary, the physician should not bind himself to any definite time, but should let it be determined by results.

Exercise.—When at length under the influence of enforced rest secondary congestions have been lessened or removed and the heart has regained a sufficient degree of strength, the patient may be permitted to gradually resume exercise. At first he may walk slowly and for a brief period about his room, care always being observed to avoid such a length of walk as causes fatigue, or such sudden efforts as produce shortness of breath and palpitation. By degrees the walks may be extended until the patient is able to leave the house and stroll leisurely in the open air. He must not, however, ascend stairs or hills until by proper exercise on the level his heart has grown equal to the effort. Apropos of hill-climbing, a word may be said of the Terrain Cure, or Oertel's plan of having patients with weak hearts develop cardiac power by ascending gentle acclivities. It consists in having a patient walk slowly up a gentle incline at such a pace as does not occasion dyspnœa or consciousness of a laboured and rapid action of the heart—infallible signs of cardiac strain. Then, when an ascent of a certain grade has been mastered, a slightly more difficult slope is to be attempted and overcome in the same careful manner as before, and thus paths of greater and greater steepness are surmounted. It must always be enjoined upon the patient that he is to make these ascents with great deliberation, not permitting himself to talk during such efforts, and stopping to rest whenever his breathing grows short or his heart begins to pound. It is possible in this manner for weakened hearts to attain much greater endurance, even to develop hypertrophy. Experience has demonstrated, however, that it is particularly suited to cases of myocardial weakness rather than of valvular disease. When a mechanical impediment to the circulation exists, as in stenosis or regurgitation, hill-climbing is dangerous, and patients are very apt to overdo. Furthermore, Oertel's method has to be very carefully supervised if it is to bring about good and not ill results. Consequently, it is but little employed as compared with other modes of treatment, and for cases of uncompensated valvular lesions is rarely advocated.

The one system involving physical exertion which gives the best results, and is adapted even to most instances of uncompensated valvular disease, is that forming a part of Nauheim treatment, and which will now be described.

Resistance Exercises.—These consist of voluntary movements by the patient of flexion, extension, and rotation of the extremities and trunk, which efforts are carefully resisted by an attendant trained to the work. Not only must the attendant understand how to resist the patient's movements without constricting the part to which he applies resistance, but he must so adjust his counter-pressure to the patient's strength as to not occasion respiratory or circulatory embarrassment. He must therefore be sufficiently skilled to detect signs of distress and to judge whether too great or too slight resistance is being offered.

The indications of respiratory and circulatory embarrassment, for which the attendant is to watch, are dilatation of the nostrils and sighing or irregular breathing, increasing duskiness or pallor of the countenance, a drawn look about the mouth, yawning, perspiration, and palpitation. So soon as any of these signs are detected the movement is to be stopped and the patient's extremity slowly allowed to assume a position of rest. Then, after a sufficient period for repose, the exercises may be resumed. Patients are very apt to hold the breath while executing these movements or to hold the body rigid, thus putting forth effort with more than the limb that is being resisted. The attendant should therefore remind the patient from time to time to continue breathing, and should see to it that his pose is easy and unconstrained. Attention to these points will enable a patient to go through the series of movements without fatigue or strain. Furthermore, the same movement is *never to be made twice in succession*, and each one is to be followed by a brief pause. It is also well in some cases to allow the individual to sit and rest occasionally during the treatment. As his endurance grows, such precautions become less and less necessary, although the attendant must never allow himself to be thrown off his guard and forget to maintain close watch of the patient's condition. Many persons of considerable muscular strength are inclined to regard the exercises as too easy and to think no benefit can accrue from such gentle exertions. They consequently want to have more resistance offered; but to all such requests the attendant must turn a deaf ear.

The last injunction to be observed is to have the movements made *slowly and without jerkiness.* Unsteadiness of movement is certain to be produced if a slow movement, particularly of the arms, is too strongly resisted. The object or purpose of these exercises is not to develop the muscles, but to influence the heart and circulation; all of which is only accomplished when the various movements are executed slowly and steadily, and the counter-resistance is accurately adjusted to the patient's strength—that is, his cardiac not his muscular strength.

Finally, the operator must not grasp the patient's arm, wrist, or leg, as the case may be, for this would hinder the free play of the muscles, but he is to exert counter-pressure or resistance by placing the palmar surface of his hand or fingers against that side of the patient's limb which looks in the direction towards which the extremity is to be moved. It often conduces to steadiness of movement for the assistant to place his other hand against some other part of the limb or trunk than that to which resistance is applied. The following description gives the exercises in the order in which they are usually executed:

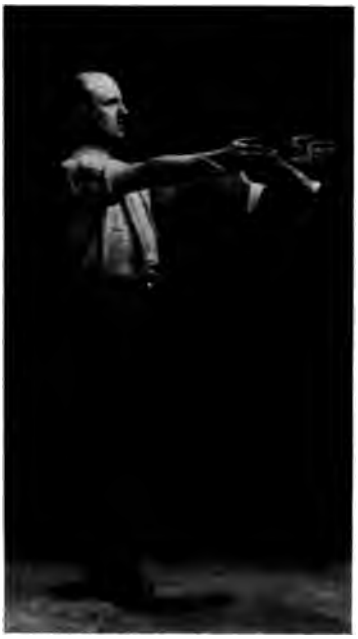


Fig. 84.

(1) The arms are extended in front of the body on a level with the shoulders and with the palms of the hands touching. They are then slowly and steadily moved outward until at a line with the front of the chest, while at the same time the attendant gently resists this horizontal movement. The attendant now changes his hands, so as to exert pressure against the palmar surface of the wrists, and the patient slowly and steadily brings his arms back to the position whence the original movement started (Fig. 84).

(2) The right arm hanging at the side, with the palm of the hand forward, the forearm is slowly flexed against counter-resist-

ance by the attendant until the fingers touch the front of the shoulder. The attendant then changes his point of pressure to the back of the arm, and the extremity is slowly returned to its former position at the side (Fig. 85).

(3) This consists of precisely the same movement, but executed by the left arm.

(4) Both arms, depending at the side, are slowly raised laterally until the thumbs meet above the head, and are then brought down to their original position, these movements being carefully resisted throughout.

(5) The patient clinches his hands in the form of a fist, but with the thumbs extended upon the ulnar surface of the index fingers. The tips of the thumbs are then gently pressed together in front of the abdomen, and, a proper degree of resistance being offered, they are thus slowly raised until the hands rest on the top of the head, after which they are slowly lowered to the original position (Figs. 86 and 87).

(6) The arms, depending at the sides, are then elevated forward and upward without bending them until they are held aloft on a line with the perpendicular axis of the body. They are next slowly allowed to resume their position at the side in the same careful manner in which they were raised. To properly resist this movement requires much practice and skill, for the reason that the hand of the attendant must be continually slipped around the patient's wrist to suit the changing attitude, first to the horizontal and then the vertical (Figs. 88 and 89).

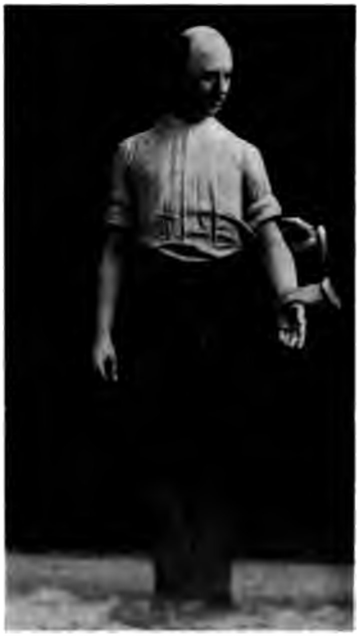


FIG. 85.

(7) Starting with the arms hanging at the side, the right arm is slowly rotated forward, upward, backward, and downward around the shoulder-joint as the pivot, and then in the reverse

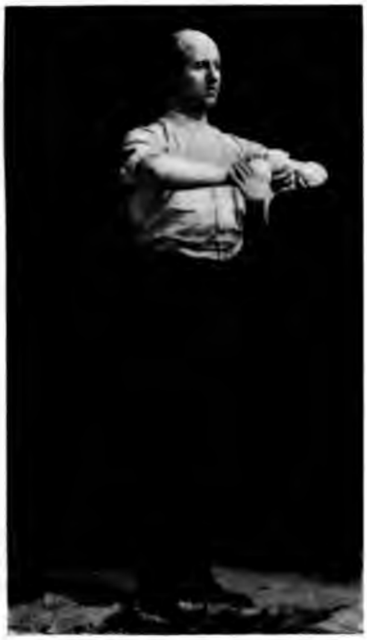


FIG. 86.

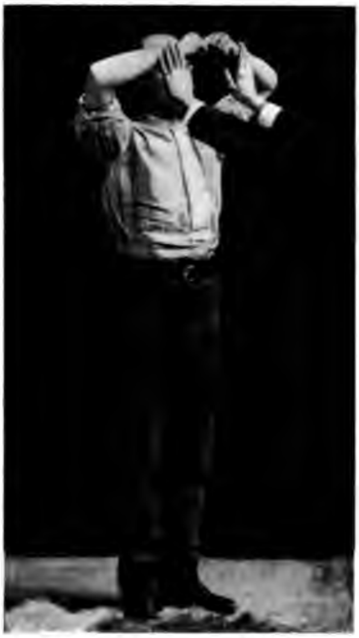


FIG. 87.

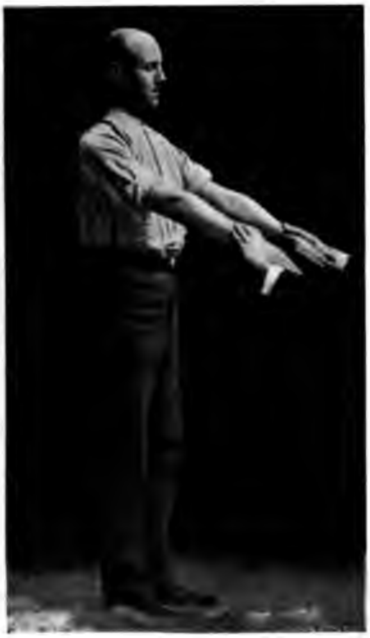


FIG. 88.

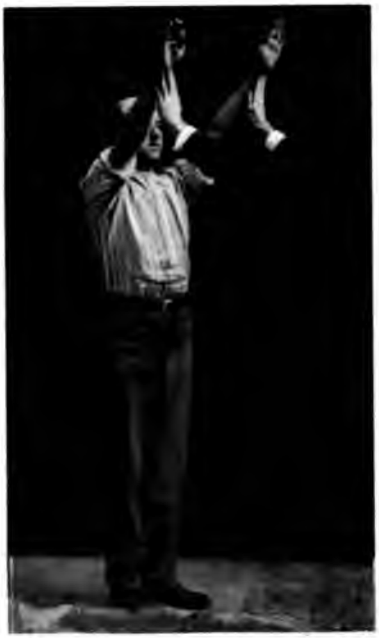


FIG. 89.

direction until the circle is completed, counter-pressure being all the time exerted by the attendant.

(8) This consists of a similar movement, executed by the left arm. These two movements are difficult both for the patient and the attendant, and should not be given to patients who are very weak or whose hearts are incapable of withstanding much exercise. Resistance to this movement is likewise extremely difficult, for the reason that the attendant has to change hands during the progress of the movement, yet without causing jerkiness or too much interference.

(9) The patient bends his body forward at the hips without flexing his knees, and then brings it back to the erect position, while the attendant, standing at his side, resists the forward movement by one hand against the upper part of the sternum and the other in the middle of the back, and the return movement of the trunk by one hand against the upper dorsal region and the other upon the epigastrium (Figs. 90 and 91).

(10) Standing with the feet firmly planted upon the floor, the patient rotates his trunk around its vertical axis, at first to the left, next to the right, and then back, so as to face forward, as before starting. The attendant resists this movement by placing one hand against the advancing shoulder and the other in the opposite axilla, and then changing his hands as the body is rotated in the opposite direction (Fig. 92).

(11) In this movement the trunk is bent laterally, first in one direction, then in the other, and lastly is brought at rest in the upright posture. To resist this flexion the attendant places one hand on the hip and the other against the side of the chest towards which the body is to be bent (Fig. 93).

(12) Both arms hanging at the sides, with the palms facing towards the thighs, are simultaneously moved backward and upward as far as possible without bending the body, and are then brought down to the sides, resistance meanwhile being carefully exerted by the attendant (Fig. 94).

(13) The patient supports himself by resting one hand on a chair, and then raises the opposite leg as far as possible in a lateral direction, while the attendant resists both the upward and the return movement (Figs. 95 and 96).

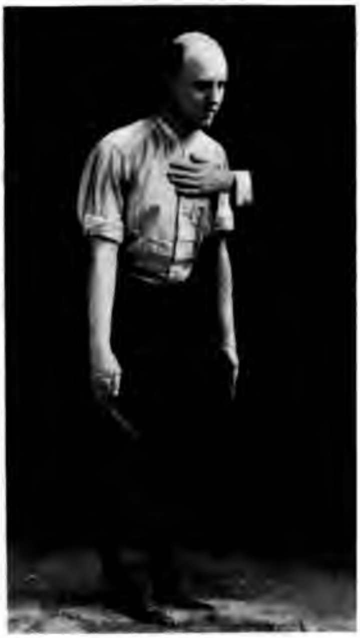


FIG. 90.

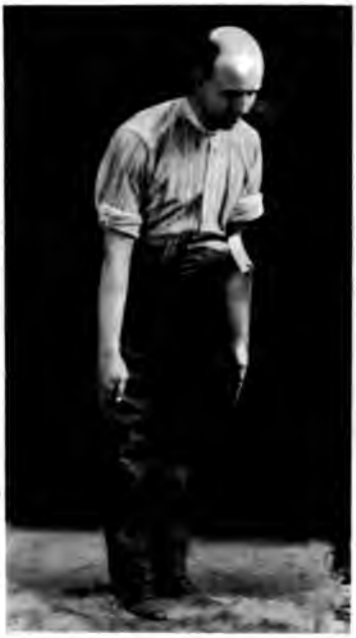


FIG. 91.

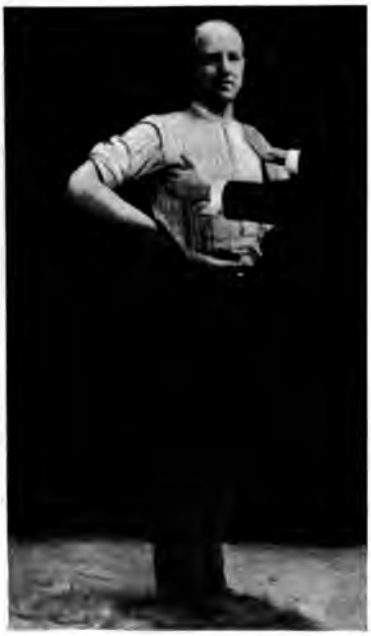


FIG. 92.

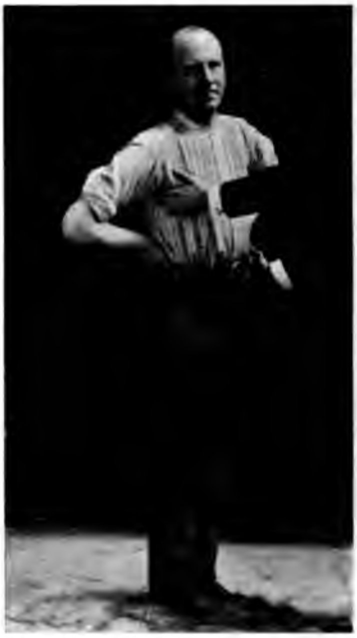


FIG. 93.

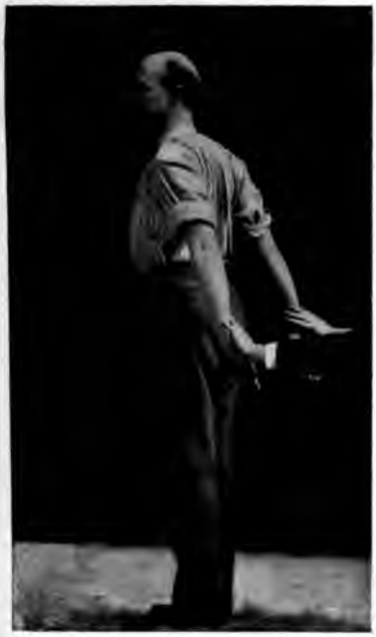


Fig. 94.

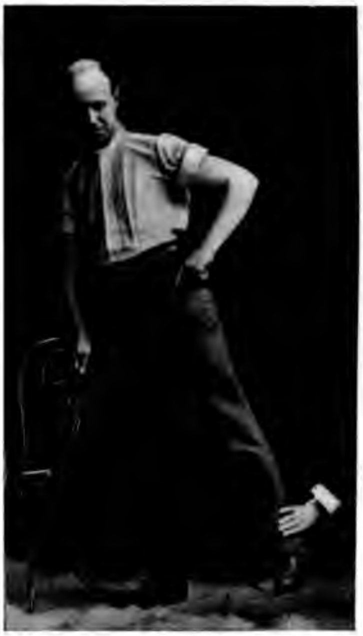


Fig. 95.

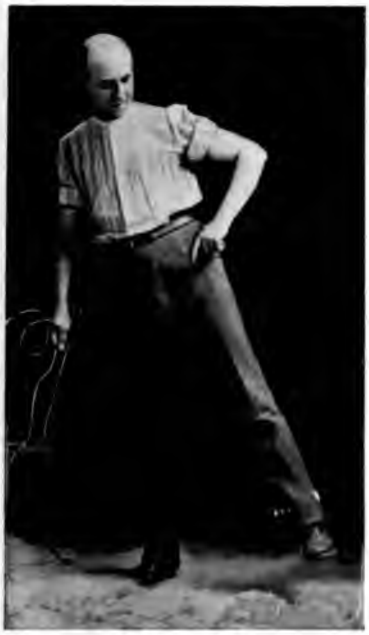


Fig. 96.

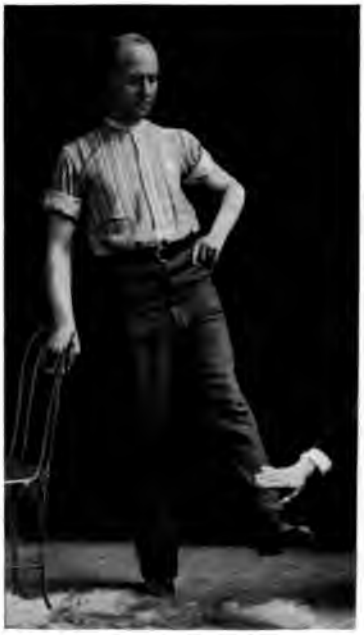


Fig. 97.

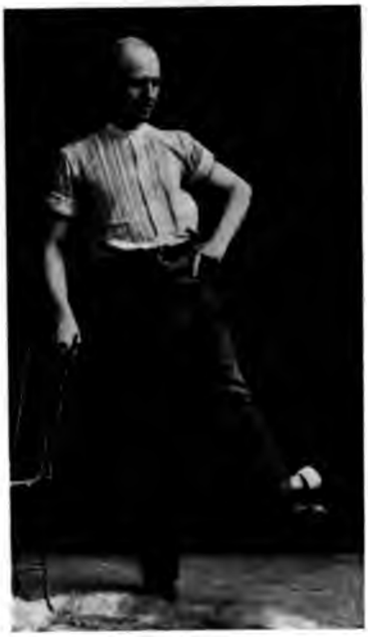


FIG. 98.

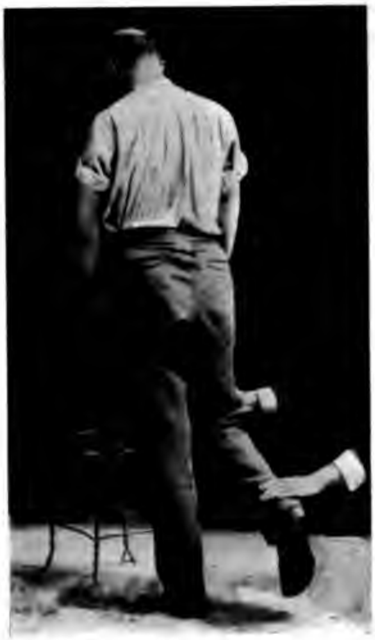


FIG. 99.

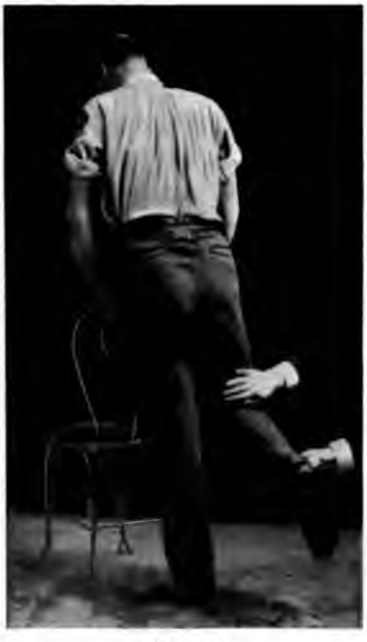


FIG. 100.

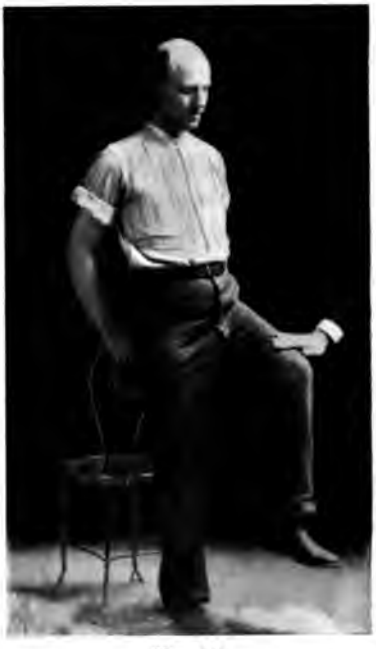


FIG. 101.

(14) This is the same movement, but done with the opposite extremity.

(15) Resting one hand on a chair, as before, the patient extends his opposite leg and thigh, but without bending his knee, as far forward and upward as possible, after which the extremity is slowly returned to its original position, resistance to both movements being offered by the attendant (Figs. 97 and 98).

(16) This is a similar effort put forth by the opposite extremity.

(17) Both hands supported on the back of a chair, one leg is flexed at the knee while resistance is offered by the attendant's hand placed at the heel. The return is resisted by the hand against the ankle just above the instep (Figs. 99 and 100).

(18) This is a corresponding movement by the other leg, resisted in the same manner.

(19) Supporting himself by the back of a chair the patient flexes his thigh at the hip, the leg hanging limp and flexed, while the attendant resists first the upward and then the downward movement (Figs. 101 and 102).

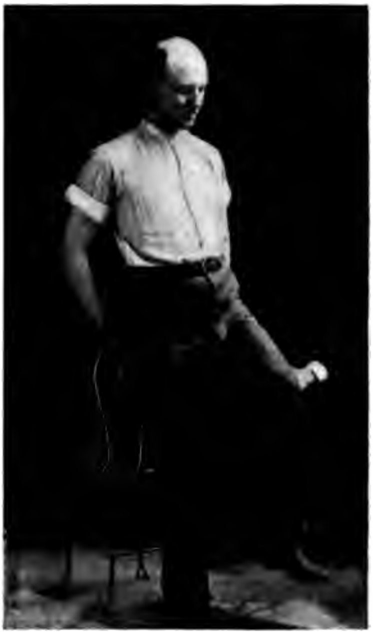


FIG. 102.

(20) This is a similar movement by the opposite thigh.

If desired, additional movements of flexion, extension, and rotation of the hands and feet may be devised. In carrying out these exercises care should be taken that movements involving the use of the same groups of muscles do not succeed each other directly, but are interrupted by exercises made by different sets of muscles. The purpose of this precaution is the avoidance of undue muscular fatigue of weak patients. Given with requisite deliberation, and with sufficient pauses for rest between movements, such a series of resistance gymnastics ordinarily takes about half an hour. If after a rest of ten to fifteen minutes the patient does not feel or evince fatigue, he may then repeat the series. They are generally given daily, an hour or more after a meal. Patients whose condition is fairly good may be allowed to perform them twice a day—that is, in the forenoon and again in the afternoon.

These exceedingly simple exercises are a powerful agent for good or a means of great harm, depending on the manner in which they are given and the condition of the heart. I do not believe they should be given to patients whose compensation is wholly gone. In this opinion I differ, I think, with Schott and Bezly Thorne, who have written so much in praise of them. If there is pronounced stenosis of an orifice, with great dilatation of the chambers back of the lesion, harm may follow their employment, the same as with digitalis incautiously given. This was sadly illustrated in one of my cases. First lessen the cardiac inadequacy by rest and other treatment, and then these movements are likely to prove highly beneficial. In more than one patient, whose enormously congested liver had refused to subside under the free and prolonged use of cathartics and heart-tonics, I have seen the organ diminish rapidly in size during the administration of resistance together with breathing exercises. It seemed as though they served to bring about an aspiration of the blood out of the engorged liver. They are far superior to massage, which seems to me to produce just the opposite effect. Massage promotes a more rapid and ampler flow of blood to the heart, while resistance movements are thought to exert their salutary effect by dilating the arterioles, and thus unloading the overburdened heart.

Nauheim Baths.—The balneological treatment of heart-disease has not received as much attention in this country as in Europe, and yet it has been growing in popularity even here. Large numbers of wealthy Americans and Englishmen annually make pilgrimages to Germany for treatment at the little resort known as Bad Nauheim, where the employment of cool saline and effervescing baths was first introduced in this class of affections. Patients of moderate means cannot afford so expensive a journey, and must either forego this treatment altogether or content themselves with the use of artificially prepared waters. For the consolation of such it may be stated that ample experience all over the world, but particularly in England, has shown that equally efficient results may be obtained in this way as at Bad Nauheim. I myself took a course of baths at that resort in the summer of 1893, and ever since my return have been employing this plan of treatment in suitable cases, and can justly claim priority in this regard over all others in this country. I have not desired to make a fad of this treatment, and therefore have not subjected as many patients to it as might have been done, but it is within bounds to say that considerably over one hundred have thus been treated by me. Some of my patients have taken the baths in Germany, generally after a course in Chicago, although one has just finished here who has previously treid the baths at Bad Nauheim. All agree in the statement that the effects with artificial waters are the same as with the natural, the chief and perhaps striking difference consisting in the more powerful effervescence of the latter, particularly in the form of the Sprudel-Strom-Bad (flowing effervescing bath). Another advantage in favour of the latter lies in the consideration that when a patient goes to Germany he leaves his cares behind him, and while there abandons himself to the one purpose of getting well. On the other hand, I have been assured that, owing to the immense number of invalids who frequent the place, patients are apt to miss the watchful care and oversight which many of them require and receive at home.

The waters of Bad Nauheim are impregnated with various chloride salts, the two to which particular virtue is attributed in their effect upon the circulation being the chlorides of sodium and calcium. In addition, the springs used for the preparation of the baths are highly charged with carbonic acid, which makes them very stimulating, particularly when used in the form of the flowing bath—that is, with a steady stream of effervescing water flowing over the body of the bather in the tub. This is comparatively rarely prescribed, being considered too exhilarating for any except those in fairly robust health. It is the rule in the employment of this balneological treatment to begin with water of a temperature of 93° to 95° F., according to the ability of the patient to react, and with water containing 1 per cent of sodium chloride and $\frac{1}{10}$ per cent of calcium chloride, but no carbonic acid, this latter being added at the end of the second week, or when a temperature of 91° to 90° F. has been attained. The duration of the first bath is from five to eight minutes, depending upon the strength and reaction of the individual. One treatment is taken daily for two or three successive days, and then comes a day of rest. This is to prevent undue depression, as is likely to be experienced when no interruption in the course of treatments occurs. The patient is required to rest, by preference lying down, after each bath, and if reaction is not good and prompt to take a warm drink of some kind and to cover up warmly. He is also required to see his medical attendant daily, or as often as the latter may elect, that the effect of the treatment may be judged of and the baths modified as his progress requires. The principle underlying the ordering of these is that the percentage of the ingredients is to be increased, the temperature lowered, and the duration lengthened until finally the chloride of sodium reaches 3 per cent, chloride of calcium 1 per cent, the temperature 87° or 85° F., and the time twenty minutes.

The rapidity with which this change can be effected depends upon the degree of objective and subjective improvement observed, but as a rule this maximum is not attained under three or it may be four weeks. In the more serious cases, or such as exhibit considerable anæmia and sluggish reaction, it is not always wise to bring the temperature below 89° or even 90° F., although the maximum in strength and duration may be the same as when lower temperatures are prescribed. It is not well to reduce the temperature more than a degree at a time, and whenever this is done the proportion of the salts is usually increased. For the most part effervescing baths are ordered at the end of ten days or two weeks, or when the higher percentages of salts have been reached; but if the patient is inclined to chilliness at a temperature that ought to produce at least a tolerable feeling of warmth, or if afterward the extremities are cold and the skin does not get into a good glow, it may be well to add the gas at an earlier period. The warmer saline baths, 95° to 92° F., are considered soothing, while the cooler effervescing ones, 89° to 85° F., are stimulating, and hence are not applicable to very weak hearts.

The direct effect of each bath should be to render the pulse slower, of better quality, and more regular if previously irregular. The area of deep-seated cardiac dulness diminishes and the heart-sounds grow stronger. Endocardial murmurs intensified by dilatation may become less loud, or if weak from cardiac asthenia, may after the bath be found to be more intense. The degree of benefit is to be determined chiefly by the size of the heart and by the character of its action. As a rule, also, the patient is conscious of a sense of well-being and of some lessening of whatever symptoms have annoyed him.

If the treatment has been judiciously ordered and overseen, the heart is found to gain in strength week by week, visceral congestions diminish, as evinced by increased diuresis, the colour of the skin grows more like that of health, and the patient gradually gains in vigour and ability to exercise without discomfort.

Just how this balneological treatment brings about improvement is still a matter of speculation and discussion, being by Schott explained on the hypothesis of increased tissue change together with a reflex stimulation of the heart which causes its slower and more powerful contractions, and with a physiological stimulation of the arterioles and capillaries by the passage of the gas and salts through the skin. By others, in particular Broadbent, the beneficial action of the baths is attributed to dilatation of the cutaneous capillaries, in consequence of which resistance to the work of the left ventricle is lessened and the transfer of blood from the venous to the arterial system is promoted. The objection urged against this explanation is, that the rate of the pulse should be increased rather than decreased, so that there must be some additional influence at work. The following is the view of Medicinalrat Groedel of Bad Nauheim: "The incontestable success which our baths have on the heart's function and the entire circulation is only to be explained if we believe in a direct action by way of the end-organs of the cutaneous nerves on the central vascular and cardiac nervous system, both trophic and motor." It may also be stated that so far as concerns the demonstrable effect of the two means of treatment, the resistance gymnastics and the baths, the results if not the action are identical in diminished size of the dilated heart and improved energy and steadiness of its contractions. Consequently it is customary at Bad Nauheim to have the patient receive both forms of treatment each day. Finally, it is usual to send the patient away at the close of a course of baths for a rest of a month to six weeks, after which he returns for another course known as the after-cure.

Inasmuch as the effect on the heart and circulation of the artificial and natural waters is identical, I will now describe how the former are prepared. The ingredients required are common salt (chloride of sodium) and chloride of calcium, bicarbonate of sodium, and compressed tablets of acid sulphate of lime. Instead of these latter, commercial hydrochloric acid may be used. The first is to be had as ordinary " butter salt " of the trade, while the calcium salt comes in iron drums holding from 600 to 800 pounds. This latter is, moreover, deliquescent, and, being corrosive, is most conveniently kept in a strong solution of definite strength. I have it kept on hand by one of the Chicago druggists, who dispenses it to my patients on my prescriptions. To begin with, the baths contain only these two ingredients, and are therefore simple brine baths. It takes from 50 to 60 gallons of water in an ordinary-sized bath-tub to immerse a person of average size up to his neck when lying in a semi-recumbent position. When the amount of water is known it is an easy matter to determine the number of pounds of salt and the number of pints of calcium-chloride solution to be added. When at length the water is to be charged with carbonic acid in addition, it is done by dissolving bicarbonate of soda, 2 pounds to each bath, and the same number of ounces of commercial muriatic acid or the compressed tablets already mentioned. The acid is so corrosive and difficult to keep without its fumes injuring the furniture of the bath-room that I now order the packages of " effervescing bath salts " manufactured for the purpose by two firms in New York city, and which are likewise kept in stock by the Chicago druggists. Each package contains 2 pounds of soda and 8 tablets and printed directions for their use. One such package is required for a single bath. These effervescing tablets possess this additional advantage over the acid, that the evolution of gas is steady and continuous. They are also, however, corrosive, and the bottom of the tub should be protected by a rubber sheet.

It is my custom to prescribe the baths in groups of three with the rest day between—that is, on every fourth day—and a course usually extends over a period of six weeks. In most cases the resistance exercises are also taken, but some hours prior to or after the bath, that the effect on the heart may be maintained. I always strive to impress patients with the importance of living as quiet and routine a life as possible, and in particular to strenuously avoid undue cardiac strain that they may not destroy the benefit expected to be derived from the treatment.

I can recall only 5 cases in which this plan of treatment seemed to do harm rather than good. Two were instances of chronic myocarditis, the hearts being very dilated and their action arrhythmic. One was a mitral lesion with œdema and other signs of rather a badly destroyed compensation; but as this patient was compelled to journey some distance each day to get his bath, it may be that the exertion thus required, and not the treatment, was responsible for the want of improvement. The remaining two were cases of serious valvular disease complicated by pericardial adhesions. In both, the engorgement of the liver became manifestly greater towards the termination than at the commencement of the course, and the treatment was discontinued. All other patients have exhibited more or less improvement, while in some instances this has been most gratifying both to the patient and myself.

I am very positive in my belief that this treatment should not be given to persons whose compensation is wholly lost, or indeed seriously broken, and therefore the consideration of this measure has been introduced in this portion of the present chapter. I have just finished giving a course of 30 baths to a lady with a pure mitral stenosis who, when she began, gave indications of failing, or at least threatened, compensation. The second sound at the heart's apex was wanting, there being only a presystolic murmur and sharp first sound. She complained of much ill-defined discomfort at the heart, and the pulse was rapid and exceedingly feeble. Before the course was completed the second sound had returned at the apex, the area of cardiac dulness was distinctly smaller, and the pulse was slower and of greater volume. She declared she felt perfectly well. In this instance, as is often my habit, I ordered the frequent use of a laxative water, and for a time 5-drop doses of fat-free tincture of digitalis thrice, then twice, and at last but once daily. I do this because it has seemed to me that in this way I have secured more lasting results.

Contra-indications to the employment of this balneological treatment are the following: Aortic aneurysm, pronounced and extensive arteriosclerosis, and, in my opinion, all cases manifesting marked signs of cardiac inadequacy, such as ascites and considerable dropsy with a greatly distended and feeble heart, and cases complicated by extensive mediastinopericardial adhesions. Chronic renal disease does not contra-indicate the treatment unless, of course, it has led to too pronounced a degree of cardiac incompetence. Lastly, it may be stated that if the pulse does not grow of better quality after the bath, but, on the contrary, is observed to become less full and strong, the treatment will not produce beneficial results and would better be discontinued.

Diet.—This is a matter requiring in this stage of valvular heart-disease very careful attention, yet concerning which notions are for the most part woefully vague and inaccurate. Physiological chemistry has not yet worked out the changes taking place in the digestive process as a result of disease. We know that the passive congestion of the abdominal organs produced by uncompensated cardiac disease leads to a chronic catarrh of the stomach (Einhorn), with diminution and even disappearance of the hydrochloric acid (Hüfler and Jorn), which, with its impaired motility, may seriously derange its function. Reasoning by analogy, we may also assume that the pancreatic and hepatic secretions are likewise altered in quantity and quality, or that if not secreted in less amounts they are poured into the duodenum in diminished quantity in consequence of catarrhal obstruction to their outflow. Just what modification in the character of the pancreatic juice takes place we do not know, yet clinical observation seems to warrant the inference that the amylopsin and fat-splitting ferment are more unfavourably influenced than is the proteolytic ferment.

Furthermore, in consequence of sluggish circulation in the veins that carry blood to the portal system, the bile is absorbed slowly from the intestine, and when secreted is watery and poor in mineral constituents. Although the secretion of bile is but a minor function of the liver, still a deterioration in its quality and diminution in its quantity must exert a baneful influence upon intestinal digestion. These theoretic considerations are borne out by clinical observations, for cardiac patients are very prone to gaseous distention of the stomach and intestines and to eructations and other indications of fermentation of the ingesta. The fatty acids thus engendered occasion still further irritation of the stomach and establish a vicious circle which augments the evils primarily attributable to disturbed circulation.

This is not the only aspect of the case. There is alteration in the metabolic processes incident to derangement in the blood-supply to the digestive and other viscera, while toxines are locally developed which either must be destroyed in the engorged and functionally impaired liver or must pass through into the general blood-stream and exert their noxious effects upon the heart and nervous system. The investigations of Husche appear to show that the excretion of urea and uric acid is altered. The retention of the former is variable, while the excretion of the latter is hindered during disturbance of compensation and increased after this has been restored.

It is not strange, in the light of the foregoing considerations, that some patients are greatly disturbed by fermentative indigestion after the taking of the simplest and most easily digested food. Dyspnœa is intensified or developed, or they are distressed by palpitation. Others are not conscious of local disturbance, but complain of pains and aches, muscular stiffness and cramps, nervous symptoms, such as despondency, insomnia, and frightful dreams, fidgetiness of the legs, and sundry other sensations that are so commonly attributed to uric-acid retention. It may be remarked here, however, that Dr. Wessner's researches appear to show that these constitutional symptoms, as well as many others, are due not to uric acid, but to indicanuria and oxaluria. One of my patients was greatly troubled by headache, insomnia, and other nervous phenomena, and Wessner's analysis of her urine collected at the time showed an enormous excess of indican and oxalic acid. Thereupon an attempt was made to stop proteids and administer carbohydrates in the hope of relieving her distress. It was found, however, that at once she began to have so much flatulent distention of the stomach and bowels with aggravation of her dyspnœa that the non-nitrogenous diet had to be abandoned for a return to meats, etc., with all their evil consequences.

The problem of how to meet the food requirements of cardiac sufferers is a complex one and most difficult of solution when we have to do with the stage of imperfect compensation. It is quite generally agreed that in cases of heart-disease uncomplicated by renal lesions the myocardium should be supplied with a relatively large proportion of proteids. The main reason for this lies in the fact that the nitrogenous principles are tissue-forming, and hence reparative elements of the dietary, and should be in excess whenever there is a demand for increased muscular work, as is the case in cardiac affections. Moreover, meats and other foods rich in proteids do not undergo the same sort of fermentation and generate so much gas as do carbohydrates, and do not so injuriously distend the hollow abdominal viscera. They are not so bulky, and therefore, relatively to the quantity ingested, contain a far larger proportion of nutrient material. For obvious reasons persons with uncompensated valvular lesions should have their dietary definitely ordered and carefully supervised by their medical attendants.

It should be remembered that digestion and assimilation are both slow, and therefore the first rule to be laid down is *that food is not to be taken at short intervals*, but ample time allowed for the stomach to empty itself before fresh nourishment is administered. The fatty acids and other irritating products of fermentation often occasion a feeling of faintness or gnawing at the epigastrium which is mistaken for hunger and thought to indicate a need for more food. In other cases appetite is poor and patients are unable to eat heartily at the regular meal-hours, and hence the friends and even the physician get the idea that the meals must be re-enforced by milk, egg-nog, etc., midway between. I have thus known nourishment to be administered every two or three hours. Such is undoubtedly a mistake. The congested and perhaps œdematous walls of the stomach cannot by energetic peristalsis empty the chyme into the duodenum as rapidly as normal, and the conditions for decomposition being favourable, the taking of additional food before the stomach is ready for it results in serious disturbances.

Furthermore, these patients are often tormented by thirst and are permitted to pour down liquids of all kinds into the already distended and irritated viscus at irregular intervals, so that abdominal distention, eructations, flatulence, and increased dyspnœa add to the patient's distress. These considerations have induced me to set five or five and a half hours as the proper length of the interval that should elapse between the meals. I do not permit milk to be taken between times, since by being curdled in the stomach it is practically the same as solid food. When in some cases the feeling of faintness and weakness makes some intermediate nourishment unavoidable, I order a clear broth or bouillon, or weak tea containing a little cream but no sugar, etc. So simple a restriction as this has often been found to work wonders in removing the thirst and epigastric gnawing. A cupful of hot water drunk an hour previous to a meal seems to facilitate the expulsion of the stomach contents and to clear the way for the ingestion of fresh food.

The next rule is the restriction of the quantity of fluids to be taken with meals. In severe cases liquids should be limited to 6 ounces, and even in mild ones 10 ounces should be the maximum. This does not mean only water in addition to any other fluids that may have been ordered, but includes all liquids combined and consumed in addition to solid ingesta. The purpose of this restriction is to prevent undue distention of the stomach in those cases in which such distention would be likely to occasion shortness of breath or embarrassed cardiac action.

The rule is that patients are to be restricted in the amount of their solid food, for it is not rarely observed that they manifest a veritable bulimia, and if permitted to do so will overload their stomach and sorely overtax their digestive and assimilative capacity. A simple and usually sufficient guide as to the amount that may be safely consumed is to be found in the injunction that they finish each meal feeling they could eat more. *A little, well digested, is worth far more than a good deal, poorly digested.* This restriction not only lessens the danger of distending the atonic organs which it has been shown furthers decomposition, but it tends to prevent the cardiac embarrassment occasioned by repletion. Such a limitation of the patient's food must not be carried to the extent of starvation; and yet if the quality of the nourishment is judiciously selected it will often be a matter for surprise how small a bulk will suffice—nay, how it will minister to the patient's comfort.

In considering the articles of food suitable to this class of sufferers I think it best to deal with the subject in a general way rather than to attempt to make up appropriate *ménus.* Tea and coffee should be weak and contain such an amount of sugar and cream as depends upon the degree of digestive disturbance. Cocoa or broma is preferable to chocolate because containing less fat, and when made with milk is nutritious. If wine or liquor is thought advisable, it should be freely diluted with water. Buttermilk, kumyss, and malted milk make a valuable addition to the dietary, and generally agree well.

Effervescing beverages are objectionable on account of the distention they occasion; and for this and other reasons malt beverages are not advisable, unless in special cases when they are craved on account of their bitter taste or their stimulating the flagging appetite. Iced drinks and very hot fluids are not well borne, since medium temperatures are better for weak stomachs. The admissibility of soups and broths must be determined by the condition of the kidneys and the habits of the individual. When chronic nephritis exists, stock soups and meat extracts are to be forbidden, since animal extractives are irritating to the renal epithelium. It should be remembered that beef-tea and the like are stimulants and possess no real food value. Cream soups, or *purées* as they are called, are not open to the same objection and are highly nutritious. All these are fluids, however, and when taken in connection with solid food should be limited in amount, lest they blunt the appetite for what is to follow and create a feeling of repletion.

Carbohydrates should be allowed but sparingly because of the following considerations: In the first place they readily undergo fermentation and occasion flatulent distention of the stomach and bowels; while in the second place they are so readily oxidized that they appropriate the oxygen necessary for the utilization of the nitrogenous principles of the dietary.

Nevertheless, sugars and starches cannot be withheld entirely, and hence they must be in the least objectionable forms. To diminish their tendency to fermentation the latter should be so thoroughly subjected to heat in cooking that the starch granules become converted into grape-sugar. Toast, zwieback, light crackers, and "pulled bread" and muffins or tea biscuits made with baking-powder are preferable to bread which has been raised by means of yeast and is often imperfectly baked. If potatoes are allowed, they should be baked and mealy, and even cooked in this way they should not be taken in unlimited amounts. Rice when well boiled may be also permitted in restricted quantity, but sweet potatoes, cereals, and the multifarious combinations of flour, butter, and sugar, whether with or without eggs and milk, known as cake, griddle-cakes, etc., are inadmissible.

Most desserts, and in particular sweetmeats, confections, preserved and canned fruits, are to be allowed only to those patients who can dispose of such articles without annoying flatulence. Fruits are best in their natural state, and even then should be ripe and fresh. Apples are particularly good because of their relatively large percentage of nucleo-albumin, and when baked are often better tolerated than when uncooked. Pineapple has always seemed to me a particularly desirable fruit because containing a natural digestive ferment of great efficiency. As a general thing I regard it as better for cardiopaths to take fruits at the close rather than at the beginning of a meal, as they do not blunt the appetite nor create so much gas.

Most of the fresh vegetables are valuable additions to the dietary, either because rich in proteids and other nutritive principles, or on account of their serving as relishes and containing various salts essential to the organism. Peas, lentils, string-beans, and spinach are said to be relatively rich in iron-forming principles. Tomatoes, cabbage, cauliflower, turnips, and kindred varieties are apt to disagree, but if well borne may be permitted. Asparagus, when not contra-indicated by renal disease, celery, lettuce, greens of various kinds, and young onions are allowable, while cucumbers, tender radishes, and olives may be left to individual desire and ability to tolerate without distress. Mushrooms are very rich in proteids, and for renal patients supply a form of nitrogenous food that is not open to objection as is animal food with its extractives. Beets are rich in sugar, as is corn, and are likely to occasion flatulence.

Of foods rich in proteids beef and pork head the list, but perhaps are not so easily digested as are veal, lamb, and mutton, which are excellent when not too fat. All meats should be roasted, broiled, or stewed, not fried; but however prepared, they should be as free from gravy as possible and ought to be so well done as to have destroyed the germs of decomposition through whose action during the time of hanging the meat becomes tender. Fowl and game-birds form a capital adjunct to the heavier meats, as also do fish and most kinds of shell-fish, particularly oysters when raw.

Some of the salted fish and meats when not too rich provide appetizing and nutritious dishes. Canned salmon, sausages, etc., are too rich in oil and fat, and are apt to cause eructations, whereas fresh tripe is said to be easy of digestion. Cheese is highly nutritious, and when known not to occasion constipation or distress may be allowed. This is especially the case with cottage and cream cheese. This article of food should not be taken when cooked. Eggs are very digestible and form a valuable addition to the dietary of this class of invalids.

Years of experience in the feeding of cardiopaths has convinced me that their food should be plain and that where more than ordinary indigestion exists the *ménu* should not be elaborate. It has seemed to me an excellent plan in some instances to separate the carbohydrates from the animal foods—that is, to give them by themselves. Then at the meals composed chiefly of animal food only vegetables or relishes, such as asparagus, lettuce, or celery, are allowed in addition. In this manner has been prevented much of the putrefactive decomposition of meats which engender the distressing symptoms of indicanuria and oxaluria.

In conclusion, a few words may not be amiss concerning an absolute milk diet in cases of cardiac inadequacy. It has been highly recommended in conjunction with absolute physical rest as an excellent means of restoring compensation when this is threatened. It acts probably by lowering arterial tension and lessening or removing the evils of the defective digestion of solid food, since milk alone acts as an intestinal antiseptic. Furthermore, by virtue of its sugar, milk often exerts a pronounced diuretic action, and thus aids in the removal of minor degrees of dropsy. When administered as the exclusive article of diet, it is best given blood-warm and in moderate amounts at regular intervals—e. g., a cupful every two hours. It sometimes agrees best when diluted with an alkaline water, as Vichy or Seltzer water. Such a diet should not be maintained indefinitely, and in most instances patients begin to plead for more substantial nourishment at the end of two or three days. It should be persisted in, however, until the results aimed at have been reached, when a gradual return to solid food is to be made.

Clothing, Habits, Occupation.—What has been said in preceding pages under these heads applies with still greater force to subjects of valvular mischief when their compensation is imperfect. The influence of these factors is subsidiary to those that have just been considered, and yet these matters are by no means unimportant. A too tight corset or waistcoat may so hamper thoracic movements and so impinge on the distended right or left ventricle as to frustrate all attempts to reinstate compensation by digitalis, resistance exercises, baths, etc. Constriction of the chest and abdomen is therefore to be sedulously guarded against.

The immoderate use of tobacco will assuredly prove depressing to the heart-walls we are striving to strengthen. Alcoholic excess, even though intoxication does not result, injures an uncompensated heart by causing excitation and exhaustion of the cardiac muscle-fibres. Sexual excess, perhaps even very moderate indulgence of this kind, may maintain or increase the very dilatation we are endeavouring to overcome. It should therefore be strictly forbidden until such time as the heart-power shall have been reinstated.

Occupation of all kinds, particularly such as involve even the lightest possible physical effort, and exciting, long-continued brain work, must be laid aside while compensation is defective. Unfortunately, the circumstances of the patient do not always admit of a rigid enforcement of this injunction. When this is the case, the danger of injury from his occupation must be explicitly stated, that the work may be performed in the easiest possible manner. It is always well to furnish such explanation, together with a warning, that in the event of damage resulting from a continuance of the employment the physician may not be held responsible for the failure of treatment. Only by attention to details can the medical man hope to turn what threatens to prove a disastrous defeat into a brilliant and it may even be an unexpected victory. It is precisely in this class of cases that modern cardiotherapy obtains its most signal and astonishing triumphs. There is no other class of cases which so amply rewards intelligent and painstaking management.

CHAPTER XVIII

THE TREATMENT OF VALVULAR HEART-DISEASE

(Concluded)

III. COMPENSATION LOST

In some cases of valvular disease that have reached this stage, restoration of their compensation is impossible and the physician can do no more than mitigate the patient's sufferings or add a few weeks or months to his life. In other cases skilful management may so assist the heart in its struggle that it is able to overcome the obstacles in the way of the circulation and regain a measure of its former adequacy. The weapons with which to aid Nature are at the command of all, but the knowledge how to make them most effective is possessed by only a few. Digitalis is the weapon on which chief reliance is to be placed. It has its congeners, which are sometimes of greater service because of some differences in their action—e. g., strophanthus, which exerts less constricting effect on the arterioles. It is safe to say, however, that therapeutists of greatest experience place more reliance on digitalis than any other remedy of its class, and that as a clinician grows in experience in the treatment of this group of lesions he generally comes to employ this drug more and more often, and strophanthus and similar remedies with decreasing frequency.

It is not alone necessary to have knowledge of its mode of action; one must also understand the indications and contra-indications for its use. The skilled hunter will procure more game with an expenditure of less ammunition than will an untrained sportsman. So likewise with this great remedy. The experienced and skilled clinician will accomplish more oftentimes with small doses than can he who is not trained to recognise the conditions that do or do not call for its administration. Inexperienced practitioners are apt to think they must order digitalis so soon as called on to treat a case of valvular disease with ruptured compensation, no matter how great the visceral congestion or extensive the œdema. Indeed, the presence of dropsy is generally considered the indication for digitalis, and hence this drug is prescribed as the sovereign remedy; when this fails, the case is considered hopeless. As a general proposition it is true; but in many cases the giving of digitalis at first is analogous to whipping a horse that cannot draw his load up hill. He fails, not because of lack of effort, but because his load is beyond his strength. Lighten his load, and the poor beast will surmount the hill without faltering. The crippled heart fails in its labours, as a rule, because its task has become too great, and not from weakness inherent in its myocardium. It has become like a jaded horse, exhausted yet willing still, which may respond for a time to whip and spur, but will die in the attempt.

Overdilatation—that is, overdistention—of the cardiac cavities renders the heart incapable of responding to a drug which slows the organ by prolonging diastole and thus favouring a better filling of its chambers. The heart is already filled to its utmost and fails to contract adequately because of abnormal endocardial blood-pressure. Even under the stimulus of digitalis its walls cannot cope successfully with its contents. As a matter of fact, the drug only intensifies its embarrassment. *The stasis within the organ must first be relieved, after which digitalis may be administered with satisfactory results.* This may be done by bloodletting or by catharsis; 12 to 16 ounces of blood may be drawn from the arm, or wet cups or leeches may be applied to the præcordia. I prefer, and have usually employed, hydragogue cathartics, because they lessen directly the hepatic stasis that coexists with the cardiac distention and forms another of the conditions acting as a barrier to the action of digitalis. The following is a case in point:

April 17, 1895, I was asked to treat Miss T., aged forty-four, who had been ill with heart-disease for about six weeks and had been given up by a number of doctors as a hopeless case. In fact, the last physician had declared nothing could be done for her, had acted on this belief, prescribed only some codeine to relieve her cough, and had gone his way. The lady gave a history of articular rheumatism twenty-five years before, since which time she had been short of breath. Her present symptoms, however, dated from about six weeks back, yet could not be traced to any special exciting cause. A pronounced aggravation of her condition had followed the exertion on the previous Sunday of dressing and going downstairs to dinner.

I found her sitting up in bed on account of dyspnœa and cough which had prevented sleep for several nights. Her colour was a peculiar bluish-yellow or slightly greenish hue, and anasarca was extreme, extending to the trunk, and including the upper extremities. The radial pulses were so flickering that the heart-rate had to be counted by auscultation. As nearly as I could determine, owing to the great arrhythmia, the heart was beating between 160 and 170 times a minute. There was no cardiac impulse perceptible. Both absolute and relative dulness was enormously increased, the latter reaching from 1½ inch to the right of the right sternal margin very nearly to the left anterior axillary line. Heart-sounds were feeble, the first being partly obscured by a murmur that seemed to possess an indistinct presystolic portion; the pulmonic sound was very accentuated and the corresponding aortic was weak. The bases of both lungs, particularly the right, showed dulness that did not shift, and numerous fine moist râles. The firm rounded border of the liver was palpable three fingerbreadths below the costal arch, and the abdomen yielded signs of ascites. The urine showed nothing more than the usual changes of congestion.

The patient's distress was pitiful, unable to eat or sleep, holding herself upright in bed without even the support of pillows, labouring for air, coughing, and expectorating bloody, frothy mucus. The diagnosis was plain—mitral disease of rheumatic origin, which at last had broken down and led to this enormous dilatation of the heart, hypostatic pulmonary congestion, hepatic engorgement, œdema, and ascites. There were no serious complications, and yet it was very questionable whether or not the heart-walls would ever recover from the enormous strain to which they were subjected. To the family I expressed a guarded opinion as to the result of treatment, yet encouraged the sufferer to hope for recovery, that she might summon courage from hope to endure the vigorous onslaughts on her œdema that would have to be made. It seemed to me useless to prescribe digitalis with the heart in that state; so I resolved to sustain it with strychnine, which, owing to the fact that she could not afford a nurse, was given by mouth, $\frac{1}{30}$ every three hours during the day, while each evening I injected the same dose hypodermically, together with $\frac{1}{8}$ of morphine and $\frac{1}{150}$ of atropine, to induce sleep and lessen the cough. Then as a package of symphorol had been given me for trial, I decided to try its effect on the dropsy. A cathartic was also administered, but not a very powerful one. The diuretic failed absolutely, and Merck's diuretin was tried with the same want of success. Then without changing the strychnine and evening dose of morphine it was decided to make use of purgation—good, vigorous purgation of the old-fashioned sort.

The patient's strength, by the way, had increased appreciably, although the œdema had only grown somewhat less hard. She was also able to take a little more nourishment, consisting of milk and eggs. Accordingly compound jalap powder in teaspoonful doses was administered until a large number of copious watery stools were secured. Indeed at one of my visits—the first, I believe, after the powder had been ordered—she was found sitting on the night-stool, and in response to my queries concerning the effect of treatment, stated she had been sitting there for two hours, preferring that to the effort and fatigue of changing from bed to the stool and back again every few minutes. The influence on the heart and circulation was wonderful and gratifying. First her cough and dyspnœa subsided and the sputum lost its bloody character; the abdomen softened down, and the œdema left the arms and flanks; the pulse grew a trifle less rapid, but appreciably more waves reached the wrist; cardiac dulness became rather less extensive also. Then I decided to let up a little on the purgation and to administer infusion of digitalis carefully prepared from English leaves, strychnine and morphine to be continued as before. The results were almost magical; the heart quieted down and daily grew in regularity and strength; dropsy disappeared entirely, and the patient, free from cough, was able to lie down with ease and enjoy a comfortable night's sleep. The morphine was discontinued, but the strychnine was left undisturbed.

May 1st the patient was turned over to Dr. Houston, who resided in the neighbourhood, and by him reports of her progress were made to me. The last time I saw the patient, about June 1st, she was sitting dressed on a sofa, without the faintest trace of œdema, the pulse 80, perfectly regular, the heart of nearly normal size, and with a loud systolic apex-murmur. Compensation had become re-established for the time. A peculiar and interesting feature of this case now developed; the patient became quiet, morose, and melancholy, very unlike her cheerful, patient self during the height of her peril. This change of disposition Dr. Houston found was due to digitalis and disappeared with cessation of the drug. This patient removed during that same summer to Moline, and there, her old symptoms of asystolism returning, she died in February, 1896.

In this case the treatment, though successful, was severe, and under similar conditions I would now advise removing the ascites by aspiration, which, by more quickly relieving intra-abdominal pressure, would permit the earlier employment of digitalis. In most cases of the kind, moreover, diuretin accomplishes the removal of the dropsy. Its failure in this instance was owing probably to the extreme renal stasis, which should first have been lessened. I have seen remarkable results follow its use in cases of cardiac dropsy in which stasis was less pronounced.

A. C., a tall, slender girl of fourteen years, was seen in consultation with the late Dr. E. M. Hale, December 14, 1891, because of heart-disease and increasing dropsy. There was a history of chorea at eight years of age and again in August, 1891, since which latter attack she had not been well. Œdema had gradually appeared and increased in spite of treatment by her home physician; hence she had been brought to Chicago and placed in charge of Dr. Hale. A urine analysis of December 9th by Dr. C. Mitchell had shown a fourteen-hour quantity of 13 ounces, specific gravity 1.027, acid, urea stated as reduced to 75 per cent of normal, a small amount of albumin and hyaline and granular casts. The symptoms were the usual ones of dyspnœa, weakness, and swelling of the lower extremities, anorexia, and constipation.

The patient lay on a couch semi-recumbent, evincing plain signs of venous stasis. The lower extremities were moderately œdematous, and the external jugulars were distended, but not pulsating. There was noticeable prominence of the præcordium and great distention of the abdomen, which yielded signs of free fluid, the liver being palpable three finger-breadths below the costal arch. The heart was greatly increased in size, the apex-beat being in the sixth left interspace outside the nipple, and there was a loud mitral systolic and short presystolic murmur. My notes do not state the pulse-rate, but mention that the pulse was regular, rapid, and small. Signs of the heart being immovably fixed in position were discovered at a much later period.

In view of the fact that digitalis had been taken without favourably influencing the dropsy, it was decided to put her on 90 grains of diuretin in twenty-four hours and administer a moderate dose of calomel. Two days subsequently the urine was reported to be 81 ounces and to contain a trace of albumin but no casts. This wilful little miss then began to be so annoyed by the frequent micturition that she refused any longer to take diuretin, and this was stopped. It was replaced by heart-tonics, first digitalis, and then $\frac{1}{10}$ grain of convallamarin thrice daily. Her improvement was so rapid that after about two weeks she was taken to her Iowa home in care of a trained nurse, by whom orders as to diet and exercise were strictly enforced. Uninterrupted gain was made until February, when the patient wearied of her restraint and my services were dispensed with. The following October I learned that she was riding horseback, playing lawn-tennis, and bicycling, but " her lips looked blue." Five years now elapsed before I again saw her, but I learned meanwhile that she had another breakdown similar to her first one, her recovery being very slow, and requiring severe purgation.

My next examination of this patient was in the early fall of 1896, at which time she was attending a young ladies' school in one of our suburbs. The heart was very greatly enlarged; its apex-beat was immovable in the sixth left interspace well towards the anterior axillary line; and mitral regurgitation appeared to be the predominant lesion. Hepatic engorgement was considerable and cardiac embarrassment was evident upon exertion. She was resorting occasionally to a few drops of strophanthus, but no cathartics.

She was induced to take a laxative water at regular intervals, which speedily lessened the visceral congestion. She also consented to try the efficacy of a course of baths *à la Nauheim* supplemented by resistance gymnastics. The results were gratifying, the dilatation of the right heart being appreciably reduced and the cardiac tone in general being greatly improved for the next two years, as she subsequently stated. She passed the winter of 1898–'99 at a young ladies' school near Philadelphia, and was in tolerable health until April, 1899, when she had an attack of acute rheumatism. This resulted in an acute pericarditis with effusion, which left her very feeble for the ensuing two months, much of that time being spent in a wheel-chair at Atlantic City. I had the opportunity of examining her in June, when the heart was found enormously enlarged, with an intense systolic apex-murmur, and also a loud diastolic one immediately following the second sound. This murmur was of maximum intensity at and about the apex, and was evidently mitral, produced by the inrush of blood into the dilated left ventricle during the beginning of its diastole. The action of the heart was unsteady, the abdominal organs were much engorged, and the patient was pale and weak. She was put on strophanthus, strychnine, and iron, and went on to her home. Her condition improved during that summer, yet remembering the benefit obtained by the Nauheim treatment in 1896, she returned for another course in October, 1899.

My notes record that the apex-impulse was broad and heaving in the sixth and seventh interspaces 5 inches to left of the sternum; absolute and relative dulness greatly increased, the latter extending nearly to the left anterior axillary line, and at the right to 1½ inch to the right of the breastbone. The apex was fixed in position, and a double murmur was still present in the mitral area, both very intense. The external jugulars were full and the liver palpable. Signs of insufficiency of the aortic valves were diligently sought for, but in vain. The mitral valves alone were involved. The enormous hypertrophy and dilatation of the left ventricle were due to the exo-pericardial adhesions acting in conjunction with the mitral disease. Baths and exercises were begun October 19th, and November 1st it was recorded that the left external jugular was turgid. The patient, contrary to instructions, was ascending too many stairs and eating too heavily. Catharsis and great care in the matter of exercising reduced the turgescence of the veins and liver for a while. On November 23d it was again recorded that the jugulars were full and the lips blue. This was attributed to her having hurried in dressing and having walked against a strong cold wind. The venous congestion again diminished, although not entirely. At the close of the course of treatments, which had extended through six weeks, it was noted that the left heart had not diminished in size at all. The enlargement of the right heart was less, however, the action of the heart less easily disturbed, and the patient felt stronger, having lost the weakness, perspirations, and sensation as if the "heart was trembling," symptoms of which she spoke at the date of commencing the treatment. The winter following she felt better than ever before.

Since the above was written this patient suffered a final breakdown, and died under my care in May, 1901. Her last symptoms have been described in the chapter on Mitral Regurgitation.

March 18, 1896, I was requested to see Mrs. F., aged thirty-four years, who had been dropsical for several months. The patient had had inflammatory rheumatism at the age of twelve or thirteen and again at thirty, soon after the birth of her second child, but had not been aware of cardiac symptoms until September 10, 1895. She had then been aroused in the night by a violent attack of palpitation; and a few weeks subsequently had suddenly developed headache, paralysis of the right side of the face, and partial left hemiplegia. This had gradually improved, leaving behind a paralysis of the extensors of the left arm and contracture of the fourth and fifth fingers. After this attack dropsy had gradually come on, and had resisted treatment by several local homœopathic physicians. For six weeks prior to my seeing her she had been unable to lie down, and had only slept by sitting with her arms supported on a table in front of her. An enormous œdema extended as high as the waist and involved also the left or paralyzed arm. The greatly distended abdomen yielded fluctuation and percussion evidence of free fluid. The liver was made out as greatly enlarged, and there were signs also of fluid in the right pleural cavity. The pulse in the right radial was very arrhythmic, small, and accelerated; the left radial pulse was, and it may be remarked still is, smaller and feebler than the right. The external jugulars were a good deal distended but did not pulsate. The apex-beat was in the seventh and eighth intercostal spaces close to the left anterior axillary line. It was at first thought to be pushed over by the right-sided hydrothorax, as the fluid in the pleural cavity was thought to be. The first heart-sound was audible in spite of a loud systolic murmur which was transmitted around to the back; the pulmonic second sound was much intensified. With exception of the shifting dulness at the right base, the lungs were negative. The urine was scanty, but gave no evidence of renal disease apart from congestion.

This case was thought to be merely one of mitral regurgitation in the stage of destroyed compensation. A year later, however, well-marked retraction of the tenth and eleventh left intercostal spaces posteriorly was discovered, Broadbent's sign of adherent pericardium, and the apex was immovable. Early in April, after I took charge of the case, a quart of clear serum was withdrawn from the right pleural cavity, after which resonance and vesicular respiration returned to that side. This was after treatment had removed the dropsy, and hence this was concluded to be an old pleuritic effusion that had escaped previous recognition.

The preliminary treatment in this case, as in that of Miss T., was heroic purgation. It seemed useless to administer digitalis or diuretics until after the excessive stasis had been reduced by catharsis, and hence the patient was assured of relief if she would have the courage to bear some very severe treatment. Her response was to the effect that she would gladly endure anything that would relieve her of her suffering. Accordingly $\frac{1}{20}$ of a grain of elaterin was prescribed hourly until it began to operate. At my visit next day I learned she had taken ten of the elaterin granules and that she had vomited 11 times and purged 20. Indications of improvement were already appreciable, and as she expressed herself as ready to stand another round, the granules were ordered repeated on the following day. Their effect was immense, after which the dropsy diminished still more. To sustain her during this ordeal she received full doses of strychnine by mouth, stimulants whenever necessary, and a concentrated nourishing diet consisting largely of eggs and strong broths. By the fifth day the circulation had manifestly improved, but she felt and appeared very weak. The family was much concerned and thought the patient was never going to endure the treatment. The sufferer was undaunted, however, and I was obliged on more than one occasion to censure certain members of the family severely for talking before the patient in a way to dishearten her. Digitalis was then ordered in tablespoonful doses of the fresh infusion every four hours, and the vigour of the catharsis was abated, from 4 to 6 watery stools daily being still secured. It was not long before the patient was able to rest in bed, a thing she had not been able to do for nearly two months. The sceptics in the family circle were now convinced and ready to assist in any therapeutic measure proposed. The œdema was stubborn, yet wholly subsided in about three weeks. When this had been accomplished aspiration of the right pleural cavity was performed with the result previously stated. Digitalis was continued daily for a year, but in the form of the tincture and for the most part in doses of 15 drops three to four times a day. The change in the pulse was very gratifying, being in June, as given in my notes, only 65 and tolerably regular. The liver still remained greatly enlarged and kept showing such a tendency to increase in size and firmness whenever the bowels were not kept freely open that at length the patient was instructed to keep on hand a saturated solution of Epsom salts, and of this to take every morning such an amount as would secure several fluid evacuations. This order was strictly obeyed, and in conjunction with the cardiac tonics produced the happiest results. Before the summer was past she was walking about the house and enjoying drives. Ascending stairs was strictly forbidden, however, and was not even attempted for at least a year.

Although this patient never lost her appetite and seldom experienced indigestion, her dietary was made very simple and of a somewhat restricted quantity that there might be no chance of her overloading her stomach to the detriment of her still enormously hypertrophied and dilated heart. It consisted in the main of a small dish of cereal and cream, a soft-boiled egg, a little buttered toast, and a cup of cereal coffee for breakfast; for dinner at midday a fair-sized piece of meat, green vegetables, little or no potatoes, some bread and butter, and a simple plain dessert, as plain pudding, or some fresh fruit, water in limited quantity being the beverage; for the evening meal she generally took a little cold meat with bread and butter and cooked fruit of some kind. After a considerable time, in consequence of the development of symptoms that seemed to indicate she was getting too much meat for the limited amount of exercise, I took away the animal food in the evening and she confined herself to her favourite cereal at this meal. This patient obeyed instructions to the letter, and, owing largely to this circumstance, gained gradually in endurance and improved in colour until at the end of two years was said by friends to no longer look like an invalid. For the past four years she has taken very little medicine and has been able to attend to her household, even doing considerable work at different times. She has been allowed to go upstairs, provided she does not hurry, and has not been injured thereby. I have seen her on the average once every two weeks, yet have not always prescribed medicine, contenting myself with seeing that everything was progressing as well as could be expected. There has been very little time when she has not taken a little digitalis, usually a single daily dose of 5 or 10 drops, but now and then, when the heart has shown a disposition to greater arrhythmia or hurry, this amount has been exceeded. There have been periods of days or a few weeks when I have seen fit to order iron or strychnine, and at a few times, characterized by unusual constriction of the pulse and scantiness of urine, she has been obliged to resort to nitroglycerin, usually to the relief of the condition. In the fall of 1899 this patient contracted a bronchitis which was attended with such a degree of fever and congestion of the right lung that for a day or two I feared she would get a broncho-pneumonia. It finally yielded, however, to rest in bed, heroin and apomorphine hydrochlorate, the heart being sustained by some extra doses of strychnine and digitalis. This patient has never shown much dyspnœa, but did for a number of months suffer a good deal from fugitive pains in the left half of the thorax with areas of intercostal tenderness, while below the right scapula and passing through to the front was at times a wearing dull pain that was only mitigated by lying down. Her liver is still very large and growing, as the months go on, perceptibly thinner at its border and harder. It drops down also, being readily pushed upward. I therefore attributed her right-sided pain to the pulling of the heavy liver on its supports.

As the reader has observed, the foregoing cases are all instances of mitral disease, and two of them complicated by pericardial adhesions. They were consequently not the most promising, yet responded to treatment in a highly gratifying manner. Such is not so with cases of aortic-valve disease, as proved by the cases detailed in the chapter devoted to Aortic Regurgitation. In 1894 I had in charge a young man nineteen years of age afflicted with insufficiency of the aortic valves of rheumatic origin. Compensation was not badly ruptured to judge from his symptoms. He displayed no œdema or marked venous stasis, almost his only subjective consciousness that all was not right being shortness of breath and palpitation upon exertion. Yet the heart was dilated and the pulse notably arrhythmic. It was hoped benefit would result from a course of therapeutic baths and exercises. As a matter of fact some degree of strength appeared to be imparted to the heart, for the impulse became more defined, the sounds more distinct, and his subjective sensations less pronounced. Nevertheless, not many weeks had elapsed after the course of treatment when he suddenly had an attack of partial syncope, on account of which he was confined to bed and cardiac tonics were administered. He did not improve, and a few days later died suddenly with manifestations that strongly suggested embolism of one of the main divisions of the pulmonary artery. An autopsy was not obtained.

The Treatment of Dropsy.—When this supervenes in the downward course of valve-lesions, it is not to be regarded merely as an indication of cardiac inadequacy, but as evidence of obstruction to capillary circulation, plus anæmia and greater permeability of the capillary walls. The pressure of the transuded serum still further obstructs capillary flow and augments cardiac embarrassment. It must be removed, therefore, before the strength of the heart can be restored. In this stage of valvular disease the occurrence of dropsy is very common, and its removal is the problem first requiring solution. In some instances this is easy and only necessitates for its accomplishment the invigoration of cardiac contractions by putting the patient at rest and by administering cathartics and digitalis.

According to my experience, the infusion of digitalis exerts a more decided diuretic action than does any other preparation or any other remedy excepting diuretin. It should be freshly prepared from English leaves, as these are more reliable than the German, which are said to contain a considerable proportion of stems. The substitution of a fluid extract for the leaves in the preparation of the infusion is never to be tolerated. The addition of squills or of a potassium salt, as the citrate or acetate, is thought by some to intensify the diuretic action of the infusion, but is objectionable. Squills is likely to occasion irritation of the gastro-intestinal tract and render the stomach intolerant of the digitalis. If potash is used in conjunction it is better alone, so that either of the drugs may be increased, decreased, or withdrawn without affecting the other.

To procure its action on the kidneys the infusion should be given in full doses, ½ an ounce every four hours, and continued for several days or so long as it continues to augment the flow of urine. Its action should be closely watched, that the remedy may be stopped so soon as signs of its cumulative effect are detected. These are slowing of the heart's contractions to 60 or less, nausea, and a falling off in the amount of urine after this has first been increased. It should be remembered that some persons become nauseated by digitalis even before it has been taken long enough to produce its poisonous effects. The most trustworthy indication that the drug would best be discontinued is found in the excretion of urine. If this does not augment after digitalis has been exhibited for two or three days, even though the pulse-rate falls, there is nothing to be gained by further administration of the medicine at this time. If persevered in there is danger of cumulative action. Again, if after having been increased for a while the urine begins to diminish, digitalis is beginning to exert its toxic effect, and ought at once to be stopped. Even if these unfavourable signs do not appear I make it a rule to withdraw the infusion at the end of five days, or after the 8 ounces comprised in the pharmacopœial formula have been exhausted. The drug continues to be eliminated for a day or so longer, and hence it is not usually necessary to follow it directly by any other similarly acting agent.

It is not uncommon for digitalis to fail to remove dropsy, and when such is the case it is well to try diuretin—Knoll. This is often surprising in its action, as in one case in which after the failure of digitalis it increased the urine from a pint in twenty-four hours to 8 quarts. Even this remedy may fail, but if it is fresh and given in large doses of 90 to 120 grains a day it generally proves a powerful diuretic. It is best given in solution, 15 grains every three or four hours, yet in conjunction with digitalis smaller doses are sometimes efficient. Diuretin has a disagreeable soapy taste, and after a time may occasion nausea and even vomiting. It also loosens the bowels in some instances. Its taste and unpleasant effects may be counteracted by the addition to each dose of a drachm or two of essence of pepsin. For the knowledge of this valuable therapeutic fact I am indebted to the wife of a former patient who was compelled to take large doses of diuretin for a long time.

I have tried numerous other highly vaunted diuretic remedies, but none aside from diuretin has ever fulfilled expectations. Sugar of milk and vegetable diuretics, as apocynum cannabinum and squills, have never yielded satisfactory results. Indeed, I do not see how they can be expected to overcome dropsy when this is due to cardiac inadequacy and renal congestion. The indications are to relieve stasis and to stimulate heart action, which they cannot do. I have not employed calomel as a diuretic since I have dreaded a possible ptyalism. When used for its effect on the kidneys, it is in doses of 3 grains several times daily in conjunction with opium to restrain its action on the bowels. Administered in this manner it is said by the Germans to prove highly efficient in cases of cardiac dropsy uncomplicated by renal disease. It seems to me, however, that one is justified in resorting to so powerful an agent only when all other means have failed.

If the dropsy is so extreme that serous transudation in the abdomen or other cavities intensifies the patient's distress, it is often found that digitalis, diuretin, or caffeine, even in heroic doses, fail to increase the urine. This is due to the impossibility of securing adequate blood-pressure in the renal arteries. Stasis in the renal veins must first be lessened if one is to induce pronounced kidney action. For this reason such enormous œdema must be diminished or removed through the skin, bowels, or by mechanical means. Profuse sweating is not advisable, since hot-air baths and pilocarpine are too depressing for heart patients. We are restricted consequently to catharsis and operative procedures.

The latter include punctures or incisions of the skin of the ankles to permit the serum to drain away. This is often an efficient mode of removing obstinate dropsy, but is likely to be objected to by the patient or his friends. Great cleanliness is required to prevent inflammation of the integument. A most excellent means of securing such drainage, and one not open to the objection of possible infection, is in the use of Southey's tubes. These are tiny silver cannulas which by aid of a minute trocar can be inserted beneath the skin of the ankles, and are then secured in place by strips of adhesive plaster or by rubber bands. Hypodermic needles can be used in the same manner. The quantity of serum that will trickle away through these tubes is astonishing.

Dropsical accumulation in the serous cavities may be withdrawn by tapping, a procedure which, if slowly done, is devoid of danger of collapse. It occasions pain, and patients generally shrink from being tapped on this account. They also often urge the additional objection that it will have to be repeated, and hence in private practice it is seldom resorted to. When consent to this proceeding can be secured, the physician should not content himself merely with having thus removed the fluid. It will quickly reaccumulate unless the advantage thus gained can be held. Consequently the tapping should be followed by the administration of digitalis or diuretin, or both. It may be well also to administer an active purge, for by such measures recourse to aspiration more than once may perhaps be prevented. In the case of most private patients it will be found that they prefer active catharsis. They have had more or less experience with purgation in times past, it may be, and they naturally look upon it as less painful than being tapped.

Cathartics.—Whenever it becomes necessary to remove serous accumulations through the intestinal tract, it should be remembered that it can only be accomplished by the production of many copious watery discharges daily. It is not sufficient that the dejections are semifluid and number two or three daily; Nature often does this much by a pouring out of serum into the intestinal canal, in consequence of which the patient has several loose, even liquid, passages daily. In spite of Nature's treatment, the dropsy goes on increasing. Nature thus furnishes the indication for treatment, and fortunately we are able to aid her by the administration of hydragogue cathartics. This procedure is sometimes objected to on the ground that the cardiac sufferer is too weak to endure the depletion. As a matter of fact the patient's weakness is due to his circulatory embarrassment, and experience teaches that instead of being enfeebled to the degree a healthy individual would be by the purgation, the cardiopath actually finds he feels stronger so soon as the primary effect of the catharsis is past. This is particularly true of mitral patients to whom heroic purgation is specially beneficial. Although in the case of Mrs. F. elaterin was highly successful, still it is so drastic that nowadays I generally order a less irritating remedy. The best hydragogue is a saturated solution of sulphate of magnesia, and of this I am accustomed to prescribe a tablespoonful hourly to an adult until it begins to exert its effect. I have known patients to take as much as 4 and even 6 ounces before getting appreciable results. The efficacy of the remedy is enhanced if the patient is not allowed to follow the medicine by more than a swallow of water—just enough to remove the bitter taste of the salts. If it is complained that the drug produces a bad feeling in the stomach, this can usually be counteracted by the addition to each dose of 5 to 10 drops of essence of Jamaica ginger, which is generally found in the house. Compound jalap powder is likewise very efficient and does not prove so drastic as is supposed. Of this, a heaping teaspoonful can be safely given to an adult daily. The old-fashioned "ten ten," which is 10 grains each of calomel and jalap, was much employed by our forefathers, and is not so severe as it is thought to be. It is better, however, to give 5 grains of calomel with soda, or a blue pill of 5 or 10 grains, either remedy to be followed after eight or ten hours by ½ an ounce of Epsom or Rochelle salts. Bitartrate of potassium is also a capital drug for the removal of dropsy, and by some patients is better tolerated than is sulphate of magnesia. Glauber's salts alone is too disagreeable, but may sometimes be combined with Epsom salts to advantage. It is one of the ingredients of Carlsbad water, by the way. Carlsbad salts are often prescribed to cardiac patients, but, to be very efficient for the removal of œdema, has to be given in large doses dissolved in considerable hot water, being said to be more efficacious when administered warm. If copious watery stools are to be secured, it is best to prescribe rather concentrated remedies and not allow the intake of much water.

In case such energetic catharsis is found to weaken the patient, his strength may be sustained by strychnine, aromatic spirits of ammonia, whisky, or some other stimulant. Furthermore, the success of such treatment depends not alone upon its vigour, but also upon its persistent continuance, day after day, until the upper-hand has been gained over the dropsy. It requires judgment and courage on the part of the physician and fortitude and faith on the part of the patient. But if judiciously persevered with, it generally rewards the sufferer.

The Use of Digitalis.—When at length venous stasis has been diminished, and the cardiac cavities have been relieved, digitalis may be prescribed, and will then be found to complete the good work begun by the cathartics. The beneficial action of this remedy is generally attributed to its increasing the force of cardiac systoles, in consequence of which the arterial system becomes better filled. But, as suggested by Broadbent, a part of its beneficial influence is to be found in the greater tonicity imparted to the vessels through its vaso-constricting action. With improved vascular tone, blood-flow is hastened, and with accelerated circulation in the capillaries and lymphatics absorption of transuded serum is promoted. In addition digitalis lengthens diastole, and thus favours the emptying of the pulmonic veins, right heart, and great systemic veins, and thus counteracts the impending stagnation of the circulation. Blood-flow in the renal vessels is improved, and forthwith there is a corresponding improvement in the renal function. Accordingly, as previously stated, it is augmented diuresis that furnishes the most reliable token of the beneficial action of digitalis.

The occurrence of dropsy is a possibility in all four lesions of the left heart, but is far less often seen in destroyed compensation of disease of the semilunar than of the auriculo-ventricular valve. When, however, œdema occurs in aortic-valve lesions, it is because dilatation of the ventricle has led to relative incompetence of the mitral leaflets with back pressure in the pulmonic system, distention of the right heart, and the establishment of a condition identical with that of uncompensated primary mitral disease.

In cases, therefore, of aortic defects manifesting dropsy the indication is for the administration of cathartics and digitalis the same as in mitral disorders. I believe, however, that the latter should be administered with judgment. There are some physicians who advocate the employment of large doses of digitalis in all cases of aortic regurgitation with broken compensation. My experience leads me to agree with Broadbent when he says that a distinction should be made between cases of aortic insufficiency with œdema and those without. When loss of compensation is shown by symptoms pointing to left-ventricle feebleness rather than by œdema and back pressure consequent upon relative mitral regurgitation, then I am emphatically of the opinion that digitalis must be given with caution. It is quite possible in such cases for digitalis to increase peripheral resistance to a dangerous degree through vascular constriction. Endoventricular blood-pressure is correspondingly raised, and if the ventricular wall is very feeble, unexpected death is not a very remote contingency. In such cases, therefore, digitalis should be given cautiously, and its effects should be attentively watched.

When, on the other hand, dropsy is present, the drug may be given more freely, although it is not likely to accomplish such brilliant results as in cases of mitral disease. Indeed, it has seemed to me that in some cases of aortic insufficiency with secondary mitral leakage large doses of digitalis actually augmented pulmonary and right heart engorgement by driving more blood backward through the mitral than forward through the aortic orifice. Such certainly was the effect observed in the case narrated at the close of the chapter on Aortic Regurgitation. Œdema was not present, yet the state of the heart seemed to call for heroic doses of digitalis in the forlorn hope of lessening the dilatation of the left ventricle. Instead of doing this, however, the digitalis appeared to aggravate back pressure, and at last death came suddenly and unexpectedly.

When the mitral valve has given way in cases of aortic obstruction there is still less prospect of reinstating the ventricle by large doses of digitalis. The impediment to outflow into the arterial system is likely to cause still freer reflux into the auricle if by large doses of digitalis the physician attempts to force the ventricle beyond a certain point.

We now come to the consideration of the question whether digitalis is equally efficient in both forms of mitral disease. Broadbent is of the opinion that foxglove manifests its happiest results in mitral regurgitation, whereas in mitral constriction the medicine is not always well tolerated. This difference is due, he holds, to the fact that the narrowing of the orifice interferes with the aspiration of blood out of the lungs which follows the better emptying of the ventricle produced by digitalis. This argument applies cogently, no doubt, to cases of extreme stenosis in which the orifice is reduced to a mere buttonhole slit, for it is evident that in such an extreme condition no agent can drive or coax an adequate volume of blood past the barrier. Nevertheless, experience teaches that even in such cases digitalis is useful if properly given. This is in accord with the following consideration: Digitalis prolongs diastole, and thus affords more time for the stream pent up in the auricle to flow into the ventricle, and hence to be expelled with the next ensuing systole. Inasmuch, however, as the capacity of the left ventricle is reduced in pronounced stenosis of the mitral ring, the blood-wave thrown into the aorta is of a necessity small, and mammoth doses of digitalis are not likely to accomplish more than moderate ones. If the beneficial influence of this remedy were limited to the left ventricle, the usefulness of the drug would be limited indeed in these cases. But it acts also on the right ventricle and left auricle, and by strengthening the vigour of their contractions it enables these chambers to exert greater propulsive force. In the light of these considerations it would be a grave mistake to conclude that this remedy is of no value in mitral stenosis even when broken compensation has led to œdema and other signs of serious stasis. The combined use of depleting measures and digitalis will sometimes achieve gratifying results. Fortunately stenosis and regurgitation are often conjoined at the mitral orifice, and hence such cases are to be treated as if only insufficiency were present.

When, therefore, digitalis is to be employed for the relief of dropsy due to ruptured compensation in mitral disease, it is to be given in large and, as Balfour says, "cumulating" doses. They must, however, be carefully watched, and the remedy must be stopped so soon as signs of its toxic action are perceived. It is my habit in such cases to administer ½ an ounce of the fresh infusion every four hours for four or five days. If it is employed in this manner, and if it is withdrawn so soon as the increased diuresis first produced begins to be succeeded by a diminution in the amount of urine, there is ordinarily no danger of serious cumulative effects. If for some reason the drug does not exhibit its diuretic action, although slowing of the pulse is obtained, then the remedy should be stopped for a day or two, after which it may again be given in smaller doses.

There is no use in administering digitalis for the relief of œdema in small doses over a long time. Thus administered it will not accomplish desired results. I have frequently seen it fail when thus given, whereas subsequently prescribed in large doses during several days it has succeeded in accomplishing what it previously failed to do.

Finally, I wish to again emphasize the statement that if this unrivalled agent is to be employed for the removal of œdema it is best given as an infusion of the English leaves. The tincture, the fluid extract, or the powder, digitoxine, and the various digitalins, whether French or German, will not prove so efficient. The tincture is preferable for prolonged administration in small tonic doses. It will undoubtedly augment the flow of urine by energizing cardiac contractions, but for the removal of œdema would have to be given in doses that would be dangerous in the hands of the average medical man. This is particularly true of digitoxine and the French or crystallized digitalia. Excepting the latter, I have tried all forms of the drug, and I think I can safely assert that all can be accomplished with the reliable tincture and properly prepared effusion that can be with the other less familiarly known preparations. I well remember my experience with digitoxine. In one case it produced so powerful an effect in what was considered a safe dose, that I speedily discontinued it in alarm. In the second case, that of a middle-aged woman with mitral disease and an arrhythmic pulse, the remedy was administered cautiously and without appreciable effect one way or the other, when suddenly, almost without warning, the patient died. I could not say her death was due to the digitoxine, and yet I have always had an uncomfortable suspicion that it was. Therefore I would urge inexperienced practitioners or such as practice in the country, where they cannot keep their patients under as close scrutiny as if they were near at hand—in a hospital, for instance—to content themselves with safer preparations.

Strophanthus, convallaria majalis, adonis vernalis, erythrophlein, and barium chloride belong to the digitalis group, and therefore possess diuretic properties, convallaria having been particularly lauded by the Russians. None of them is the equal of digitalis, however, and in serious cases they are not likely to prove so reliable. I have used them as adjuncts or substitutes for foxglove when this had to be discontinued temporarily or could not be tolerated, but I have never ventured to rely on any one of them exclusively when dealing with a critical case of cardiac incompetence from valvular disease. In the young with rheumatic endocardial lesions, or in older persons whose arteries are not appreciably stiff, one need not apprehend ill effects from the vasoconstrictor effect of digitalis, while if the remedy be given in ice-water, or if the fat-free tincture be used, it will rarely disagree seriously with the stomach. When the vessels are atheromatous its effect on the arteries must be reckoned with, and then strophanthus may have to be used instead or be combined with digitalis. In these cases the latter can generally be employed successfully even for the treatment of œdema if nitroglycerin be given often enough to counteract the constriction of the arterioles.

Before concluding the subject of the administration of digitalis I wish to direct attention to the possibility of its occasioning mental symptoms that may be misunderstood and attributed to the disease instead of to its right cause. These are hallucinations and delirium. H. O. Hall has recently contributed a paper on this peculiar action of the drug, and quotes from an article thereon by Duroziez, who reported twenty instances of the kind. In this present work I have referred to the fact that in two patients whom I attended in connection with Dr. Houston a peculiar mental and emotional state developed during the prolonged administration of digitalis and disappeared after the discontinuance of the remedy. In Miss T., with mitral disease, there was a singular sort of sullen moroseness with taciturnity, while the other patient, a man with aortic insufficiency, manifested a mild delirium of a harmless kind. Hall suggests that this effect may follow the administration of even moderate doses for a considerable period, and very properly queries if it does not occur far oftener than is suspected. For my part I frankly confess that until my attention was directed to this singular effect of digitalis by Dr. Houston I had no suspicion of its possibility. There may be no danger associated with this action, but it may indicate an unusual degree of susceptibility to its influence, and that in such persons one should be especially on his guard against the cumulative action of the agent. This latter is no fancied one, and should always be kept in mind.

It is probable that digitalis poisoning occurs far more frequently than is suspected. I am painfully certain that in one instance the sudden death of one of my patients was due to digitalis, which was being administered in large doses. In this case, however, the patient disobeyed my emphatic injunction to remain absolutely quiet in bed, and fell dead while walking about, just as I had warned him he might do if he got up. Since that time it has been my custom to discontinue digitalis every fifth or sixth day in all cases in which it is being taken in considerable doses or when the patients are not under frequent observation. In this way time is allowed for the elimination of the drug. Lastly, it is said that there is less danger of a cumulative effect if a daily cathartic is taken, since it may be largely eliminated through the bowels.

Accessory Heart-tonics.—Strychnine is a valuable heart-tonic at all times, and when compensation is lost is of great value. It should not be depended on alone, but prescribed as an adjuvant to the cardiac energizers already mentioned. Administered hypodermically it is undoubtedly more rapid and powerful than by the mouth, and would best be employed in that manner. Its beneficial action is not confined to the heart, for it is a respiratory stimulant as well, tending thereby to lessen the sense of dyspnœa. Through its powerful effect as a tonic to the nervous system it induces a feeling of strength and well-being very soothing to the tired, often irritable sufferer. It certainly helps a patient endure the insomnia that so often attends his loss of compensation.

I have always been of the opinion that to display its kindly action strychnine should be given in full doses, care being had to avoid its physiological effects in toxic amounts. One thirtieth of a grain hypodermically may usually be administered every four, or in extreme cases every three hours. I have not hesitated to order that dose as often as every two hours, or even hourly in times of great peril. The objection to such doses have been previously stated. (See page 445.)

Another remedy of inestimable service when the heart threatens to fail altogether or the patient is tormented by dyspnœa, particularly at night (cardiac asthma), is morphine. If administered hypodermically and in small doses this drug proves a powerful cardiac stimulant. An eighth or a tenth of a grain thrown under the skin will arouse a flagging heart, steadying its action and improving the quality of the pulse in a manner not equalled by any other agent of which I am aware. If $\frac{1}{160}$ of atropine is combined it serves to warm up the skin by flushing the capillaries, and by deepening the respirations relieves dyspnœa. Given at bedtime, morphine will generally carry the sufferer through the night without his wonted attack of dyspnœa and depression. In some instances it acts as a hypnotic, but even when it fails of this action it allays restlessness and induces a sense of well-being that is most grateful. Larger doses are more or less depressing, and exhibited by the mouth the stimulating action of the remedy is not the same as by subcutaneous injection. I have known many a patient to tolerate morphine in this manner for weeks, and when it was at length withdrawn not to experience any special discomfort. It is not to be resorted to indiscriminately, but only in those cases in which it is necessary either to tide over a period of crisis or to promote comfort of body and repose of spirit. Of course for the relief of pain larger doses are necessary, as is likewise the case when it is desired to produce sleep.

Hypnotics.—The ultimate aim of treatment in the stage we are now considering is the restoration of circulatory equilibrium, and thereby of heart-power, and yet we should never forget that the accomplishment of our aim often depends almost as much upon attention to subordinate or accessory conditions as upon measures addressed to the heart directly. For instance, cardiopaths suffering from ruptured compensation are very apt to complain of actual insomnia or of fitful and unrefreshing sleep. This may be due to disturbed cerebral circulation, to retention of waste products, or to the generation of toxines, or to all these factors combined. So long as natural sleep is wanting, the invalid is deprived of what Shakespeare has so fitly termed "Nature's sweet restorer," and the exhaustion of the nerve-centres following prolonged wakefulness may throw the balance against recovery. Moreover, the heart itself is robbed of the rest which comes from its slower action during sleep. It is most important, consequently, to combat this distressing condition by such means as will prove harmless.

In some cases sleep follows the measures directed against visceral congestion and œdema, or is directly induced by the hypodermics of morphine administered for relief of nocturnal dyspnœa. When such is not the case, recourse should be had to hypnotic remedies as such.

Chloral hydrate is too powerful a cardiac depressor to be safely employed in uncompensated valvular disease, or indeed in any case in which the pulse is not strong and tense. On the other hand, chloralamide-Bayer is said to exert no injurious effect on the circulatory apparatus. If it affects the pulse at all, it is by accelerating it while at the same time augmenting its tension. The main drawback to this agent is its liability to occasion headache next morning and its unpleasant acrid taste. To obviate the former effect, therefore, an equal amount of potassium bromide may be added to each dose of chloralamide, while its disagreeable taste may be disguised by some palatable sirup. The remedy is perfectly safe in doses of from 15 to 40 grains, and to prove efficient ought to be given in a single full dose. A good formula is the following: Chloralamide-B. 2 grammes, spiritus frumenti 15 cubic centimetres, potassii bromidium 2 grammes, and syrupus glycyrrhizæ, q. s., ad 30 cubic centimetres. M. et sig. This dose to be taken at bedtime.

Chloralose is a hypnotic highly recommended by Balfour in his capital work The Senile Heart. Its dose is from 2 to 8 grains, best given in capsule, and is said to tend to slowing of the pulse while at the same time lowering its tension. I formerly prescribed it a good deal, but ultimately abandoned it because I found that when my lady patients took 5 grains at a single dose, or were obliged to repeat a capsule containing 2½ to 3 grains, they were apt to complain of nervousness the next morning; it is, however, safe and usually produces sleep quickly. Sulphonal and trional are both safe and efficient hypnotics for the class of cases now considered, since although they accelerate the pulse and somewhat raise arterial tension, they do not depress the heart. Paraldehyde in full medicinal doses of a drachm acts similarly to chloral hydrate as a soporific, but unlike the latter is perfectly safe even for weak hearts, being said to slow and strengthen the pulse. Its action is not so prolonged, however, and patients often object to it on account of its burning taste and persistent odour in the breath. This does not by any means complete the list of available hypnotic remedies, but comprises those that are the most efficient for cardiac sufferers. Should these not occasion sleep, and in uncompensated valvular lesions insomnia is often most intractable, recourse would better be had to morphine or some preparation of opium, of the advantages of which as a heart-tonic I have already spoken.

Rest.—Having dwelt at some length on the medicinal management of this stage of valvular heart-disease, I now desire to add a few remarks concerning the great importance of physical repose. Nothing is more essential when compensation has failed than the strict enforcement of absolute rest. There is no greater mistake, and nothing that will more surely render futile all attempts to remove dropsy, than to permit the patient to walk about. Even the moderate exertion of visiting the closet in an adjoining room will often be sufficient to maintain the venous congestion and œdema. The patient should be required to remain absolutely in bed or on a couch, and he should make use of a bed-pan. Only in those instances in which patients find it impossible to so empty the bowel or bladder should this rule find an exception. When such is the case, they may be permitted to leave their bed and sit upon a night-stool placed close at hand, or better still use an adjustable bed. The effort of raising themselves in bed will often in cases of extreme dilatation suffice to frustrate all attempts at a reduction in the size of the organ. Therefore, dropsical patients should have the necessity of physical repose clearly explained to them, and should be kept at rest until all traces of œdema have disappeared. In mitral disease this precaution will of itself often do much towards removing symptoms. In cases of uncompensated aortic insufficiency absolute repose is of the utmost importance, since a single injudicious effort may occasion paralysis of the overdistended left ventricle in diastole.

Another danger arising from exertion in cases of extreme and long-continued back pressure in the lungs is the establishment of relative pulmonary regurgitation. When this once supervenes, it is in my experience impossible to ever again restore compensation, and the end is not far distant.

Exercise.—Only after the cardiac cavities have become unloaded, and compensatory hypertrophy has begun to be restored, dare the patient be allowed to indulge in exercise. Even then exertion must be very slight at first, and the medical attendant should observe the effects of effort, and thus form an intelligent opinion of how far cardiac strength has become re-established. I make it a rule to be present the first time walking is attempted, so as to note the effect of exertion on the pulse.

Baths.—In this critical stage of cardiac incompetence I believe Nauheim baths contra-indicated, and even in the matter of bathing for the sake of cleanliness patients must content themselves with the morning sponge-bath given by the nurse. It involves too much exertion for them to bathe themselves, and particularly to get into a tub.

Receiving Visitors.—Apparently trivial influences may make for or against the restoration of heart-power. Consequently, when there is a damming back of the blood into the lungs and right heart, this latter chamber should not be additionally strained by prolonged conversation. The reason for this lies in the consideration that when words are uttered the breath is held, and that with expiratory effort air is driven out through the partially closed glottis. That this throws additional burden on the heart is plain, and is proved, moreover, by the clinical observation that cardiopaths nearly always exhibit breathlessness while talking. Therefore, these invalids should not be allowed to receive visits from friends, unless perhaps from a few who can be relied on to monopolize the conversation, and who know enough to leave so soon as the patients exhibit signs of weariness.

Diet.—This is a matter of the very greatest importance, and must not be left to the whims of the invalid or the zealous but ignorant notions of friends. The considerations and principles which obtain in cases of failing but not yet wholly lost heart-power apply with added emphasis to these, and hence the reader is referred to what has been already said.

Although the management of this stage of valvular lesions is to be conducted along definite lines, still it is and must of a necessity be largely symptomatic. There is a certain routine about it, and yet the physician must be ever alert to detect signs of danger and avert it by prompt action, and equally to take advantage of all circumstances that exert an influence for good. He should not exhaust his patient by unnecessary examinations, and yet he should look over the case daily with sufficient care to detect the earliest signs of any of the many complications to which the patient is liable. It is especially necessary to make frequent and thorough analyses of the urine, and he should insist on a record being kept of the temperature for the detection of some of the terminal infections so frequent in these cases. If he cannot restore cardiac energy, he can at least prolong life and do much to minister to the patient's comfort.

If he be so fortunate as to aid Nature in re-establishing some degree of cardiac power, the case then becomes one of the second class, and is to be managed along the lines laid down in the second portion of this chapter.

SECTION III

DISEASES OF THE MYOCARDIUM

CHAPTER XIX

ACUTE MYOCARDITIS

It is not the design in this chapter to consider acute inflammation of the myocardium in association with acute peri- or endocarditis, but the acute inflammation observed in the course of specific fevers and other acute infectious processes, and which usually exists independently of inflammation of those membranes. This form of myocarditis is described by authors as acute interstitial and acute parenchymatous myocarditis, the latter, as remarked by Osler, being regarded by some as a degenerative process.

The history of acute myocarditis is not clear until we come to the works of comparatively recent years. Suppurative myocarditis has been recognised since the earliest days of medicine, and by Galen was regarded as the disease of gladiators (Huchard). Beniveni, in the fifteenth century, discovered an abscess in the wall of the left ventricle, and in 1553 Nicolas Massa found an abscess in the right ventricle with a sinuous tract extending into and perforating the auricle.

Morgagni was familiar with myocardial inflammation, and Senac devoted a chapter to this affection.

Since the beginning of the last century the names of innumerable workers, including Corvisart, Hope, Andral, Laennec, and Stokes are linked with the history of myocarditis, but their views were more or less obscure. For the most part the changes were spoken of as a softening of the heart-muscle.

Bouillaud considered this softening as due to inflammation of both the muscle-fibres and interstitial connective tissue, and distinguished three varieties: The red, which is acute; the white or gray, which is purulent; and the yellow, which is a chronic phlegmasia.

Rokitansky distinguished acute interstitial and acute parenchymatous myocarditis, and gave an excellent description of them as he observed them in cases of typhus fever.

Virchow's studies on parenchymatous myocarditis opened a new era, for in place of the old-time changes in the consistency of the heart-muscle he described definitely recognisable microscopic and chemical changes in the structure of the muscle-fibres. He was followed, in Germany, by Stein, von Zenker, and others, while in France, Hayem did noteworthy work along the same lines. The three divisions, which Hayem made according to the duration of the process, were thought, however, to be too sharply drawn.

Among more recent German writers who have made valuable contributions to acute myocarditis as observed particularly in diphtheria, are to be found the names of Birch-Hirschfeld, Leyden, Rosenbach, and Romberg. The last-named is considered by Fraentzel to have added greatly to our knowledge on this subject, and I desire to acknowledge my indebtedness to his lucid and eminently practical exposition of the clinical features of acute myocarditis, as set forth in Ebstein's Practice.

Morbid Anatomy.—The myocardium is the muscle layer of the heart, and corresponds to the media of the arteries. It is thick in the walls of the ventricles and thinner in those of the auricles.

The lesions of myocarditis are usually most pronounced in the ventricular walls on account of the greater work thrown on these portions of the heart-muscle. The fibre of the heart-muscle is structurally between that of voluntary and involuntary muscle. The individual cells are short cylindrical bodies, containing one nucleus each. The greater portion of the protoplasm is differentiated into contractile fibrillæ which possess the optical characteristics necessary to give the appearance of striation, the fibre being thus striated in both the cross and longitudinal direction.

The myocardium possesses a very rich capillary blood-supply which is derived from the coronary arteries, and also from minute arteries opening directly from the left ventricle. Normally there is but little interstitial tissue in the myocardium, which is separated from the pericardium by a variable layer of fat, while the endocardium lies directly on the muscle layer.

Acute myocarditis is either parenchymatous or interstitial. The parenchymatous form includes the various acute degenerations of the myocardium, which are usually dependent on the presence of irritants in the circulation, such as the toxines of the infectious fevers. Cloudy swelling and granular degeneration are the most common manifestations of the process. In them the myocardium appears pale and opaque, and is soft, flabby, and easily torn.

Microscopically the fibres are swollen, their protoplasm more or less granular, and both the cross and longitudinal striations are obscured. The degeneration induces a more fragile condition of the fibres, so that they are often found ruptured or separated along the cell boundaries—a condition of fragmentation or segmentation. In these cases the rupture probably takes place *in articulo mortis.* Both these forms of degeneration are usually diffuse and not confined to any special areas.

A form of acute myocarditis which may be classed as parenchymatous, and which sometimes leads to serious results, is that which follows embolism of the coronary arteries. Infarction of the heart is followed by coagulation necrosis of the tissue involved, and is usually attended by some inflammatory infiltration of the area. Ultimately the necrotic area is replaced by scar tissue in the manner to be considered under the heading of the chronic form of the disease.

Acute interstitial myocarditis may be purulent or simple. The purulent form is usually characterized by the formation of abscesses, which may be many or few in number. On section these appear as whitish or grayish areas of softening, which are depressed below the plane of the cut. The larger abscesses may contain fluid or semi-fluid pus.

Often associated with acute endocarditis, it may be a direct extension from the disease of the endocardium. In this case the foci of suppuration are larger and less numerous than in the case of suppurative myocarditis dependent on a general pyæmia when the foci are numerous, widely scattered, and may be so small as to be barely visible to the unaided eye. Microscopically the smaller foci appear as masses of polymorphonuclear leucocytes surrounded by a zone of degenerating muscle.

Bacteria can often be demonstrated by appropriate methods. The abscesses rarely attain large size on account of the early supervention of death. They may rupture into the heart or into the pericardium, or the wall may become so weakened as to produce rupture through the entire thickness of the heart.

The simple form of acute interstitial myocarditis is a rare condition which is found in some of the infectious diseases, notably typhoid fever and diphtheria. In this the chief lesion is the infiltration of the tissue with lymphoid and plasma cells. These foci of infiltration are more numerous in the left than in the right ventricle, and are usually situated close beneath the endocardium. In these foci of infiltration there is usually considerable degeneration of the muscular tissue, which is characterized by swelling and destruction of the nuclei.

Associated with acute myocarditis are the various conditions to which the disease is secondary. Chronic myocarditis may often, though not always, be secondary to the acute form. In abscess of the heart, rupture into the circulation may cause general pyæmia and septic embolism.

Etiology.—Acute inflammation of the heart-muscle is observed in connection with such acute infectious diseases as diphtheria, typhoid and typhus fever, small-pox, scarlatina, gonorrhœa, and even articular rheumatism.

Freund has described a case of acute diffuse myocarditis of the purulent variety in a forty-eight-year-old butcher who furnished no evidence either before or after death of any other infection than that of inflammatory rheumatism. He also cites similar instances collected from the literature. Whether in cases of rheumatic arthritis there is some secondary infection that is responsible for the myocarditis, or it is the rheumatic poison itself that creates the mischief, it is impossible to decide. In the specific fevers it is the specific infection probably which gains access to the heart-muscle and there excites inflammation. It may also be that the character of the myocarditis is determined by the virulence of the micro-organism. Romberg thinks it is the intensity of the poison and not its continued action which determines the ultimate course of the inflammatory process, for "in many cases the disease of the heart-muscle reaches its highest point a considerable time after the decline of the infection, and it is not to be assumed that all this time the toxic agency continues to increase in activity."

The process may possibly be compared, he thinks, to the inflammatory reaction set up in a part whose function has been destroyed by a burn, or to the tabes dorsalis which develops as an after effect of syphilitic infection.

In May, 1900, Poynton reported a comparative study of the changes in the heart-wall in one case each of diphtheria, rheumatism, and chorea. In the first-named affection, which occurred in a child of five years, and proved fatal on the seventeenth day, the changes were those of acute parenchymatous degeneration, the muscle-fibres showing profound destruction, in some places even to the disappearance of the muscle-cells with retention of only the reticulum. Associated therewith was a cellular infiltration of the interstitial connective tissue. The endocardium and pericardium were free from disease.

In the heart of the rheumatic patient, a young man with clinical evidence of mitral disease on admission, and a day or two later of fresh pericarditis, which lesions were found after death, the muscle-fibres showed extensive fatty degeneration, but were not so profoundly disintegrated as in the case of diphtheria. There was also an exudation of cells into the connective tissue here and there around the blood-vessels.

In the case of chorea, a child that had manifested great rapidity of cardiac action, the muscle-fibres of the heart also showed more or less fatty degeneration with scattered areas of cell exudation into the interstitial connective tissue. The changes resembled those found in the heart of a rabbit that had been rendered pyæmic by the injection into the circulation of streptococci.

In commenting on the microscopic findings in the diphtheritic heart, Poynton pointed out their close similarity to those described by Mollet and Regaud, and produced by the injection of diphtheria toxines into lower animals. As to the myocardial changes in the rheumatic case Poynton was of the opinion that they were without doubt due to the rheumatic poison, and that they strongly suggested the likelihood of the rheumatic virus being a toxine of microbic origin; their clinical significance was very obvious and proved that the cardiac failure in rheumatic patients with valve defects is not always to be attributed to mechanical causes.

Whereas these observations of Poynton are not new, they are of value because contrasting and illustrating the effects on the myocardium of these two important diseases, diphtheria and rheumatic fever.

Purulent inflammation of the myocardium is found in connection with pyæmia and ulcerative endocarditis, the bacteria being brought to the heart-muscle in the blood or gaining access directly from the endocarditic affection. Septic emboli may enter a coronary artery and give rise to abscesses. The primary focus of infection may be a suppurating wound, or micrococci may effect an entrance into the system without leaving any trace of their port of entry behind.

In rare instances acute myocarditis has appeared to follow trauma, as in a case reported by Rindfleisch of a man who gave no other etiological factor than a fall from a considerable height, and striking on the left side of his chest.

Symptoms.—The observations of Romberg and Schmaltz have shown that in diphtheria the symptoms of acute myocarditis occur in from 10 to 20 per cent of all cases, and in the majority of instances appear in the second or third week of the illness, occasionally towards the end of the first, and rarely as late as the sixth or tenth week. These symptoms are the expression of diminished heart-power, and naturally, as regards intensity, depend upon the degree of the myocardial inflammation. Although for the most part they are so apparent that they cannot escape detection by a careful observer, yet there are cases in which death takes place suddenly without previously recognisable symptoms. On the other hand, according to Romberg, the symptoms of acute myocarditis, when occurring in typhoid fever, scarlatina, smallpox, gonorrhœa, and acute articular rheumatism, are less conspicuous than those of diphtheria; particularly is this true during the stage of fever. Furthermore, it is often impossible without postmortem inspection of the heart-muscle to determine whether the symptoms are not due merely to functional derangement of the heart's action.

In all cases of acute myocarditis which is not purulent it is a subject for speculation whether the weakening of cardiac energy is due to destruction of the muscular elements, or to the mechanical effect of cell exudation into the interstitial connective tissue, or depends merely upon functional depression (Romberg).

Subjective symptoms are not aways present, and if not wholly lacking are not always pronounced, and therefore close observation on the part of the medical attendant should never fail. *Pallor of the countenance* is present, and is often a striking phenomenon. Poynton mentions it in both of his cases of diphtheria and rheumatic fever, especially the former. It is due, according to Romberg, to defective filling of the cutaneous vessels, since the blood is of normal composition. *Vomiting* is another symptom sometimes of great importance, and has been dwelt on by Villy in connection with the cardiac failure of diphtheria. It may be so persistent as to suggest some abdominal disorder. There is anorexia, and the patient is often strikingly weak and listless, or instead of apathy may display restless anxiety. In a word, the patient conveys the impression of being profoundly ill.

The features of the case, however, that most strongly point to myocarditis pertain to the seat of mischief—i. e., the heart. The pulse is more frequent than normal, although as a rule its rate is not greatly accelerated, being 100 or thereabouts. Arrhythmia may or may not be present; often there is only irregularity in force and volume. Its striking characteristics are feebleness and emptiness, and as the disease progresses loss of stability as well as volume, very slight exertion being sufficient to send up the pulse-rate *out of all proportion to the degree of effort.*

Examination of the heart at this time may disclose some increase of dulness, particularly of the relative, to the left and upward, but also to the right. Yet in the beginning, sometimes even throughout the course of the malady, marked dilatation is not detected. Cardiac impulse is absent, and the sounds are notably feeble, especially the first at the apex, which is often so toneless as to be almost inaudible. In a few cases dilatation of the left ventricle reaches such a degree as to permit relative incompetence of the mitral valves, which is declared by a soft, it may be feeble systolic apex-murmur. The disturbance of the circulation may be further shown by hepatic engorgement, and in some cases patients complain of pain in the region of the liver.

The presence or absence of other signs of venous stasis, as œdema and scanty, albuminous urine, is determined by the degree of circulatory embarrassment. In some cases there is much præcordial oppression and anxiety that may amount even to pain of a dull and oppressive or poignant character. Freund lays great stress on substernal pain, and thinks it a highly significant symptom and far more pronounced than in endocarditis. In his case the patient often indicated the sternal region as the seat of his discomfort, and declared he knew he was going to die.

The course of acute myocarditis is very variable. It may set in abruptly and progress rapidly with severe symptoms, leading to death in two or three days or one or two weeks; or it may arise insidiously and be latent throughout, even up to the moment of sudden, unexpected death; or there may be alternation of periods of entire absence of subjective symptoms, with times of alarming weakness and indications of threatening dissolution.

The cases which in one sense are the most dangerous are those in which the myocarditis develops weeks after the disappearance of the diphtheria, at a time when convalescence is thought to be progressing satisfactorily, and the patient has perhaps passed out from under the care of the physician. In such the child, unconscious or uncomplaining of symptoms, plays about as usual, and one day making some extra exertion falls to the floor and expires without warning.

Romberg is of the opinion that the circulatory disturbance is not to be explained on the hypothesis of mechanical obstruction merely, the same as in cases of chronic cardiac disease, inasmuch as cyanosis, dyspnœa, and œdema are not prominent symptoms. The pallor and arterial emptiness are rather the effects of the toxines on the vaso-motor centre, of the kind shown by his and Pässler's experiments to result from acute infections. It is possible also that splanchnic neuritis, as suggested by Veronese, may be a factor, producing stasis within the great abdominal vessels, paralysis of the vagus being out of the question. Only in some such way can one account for the absence of pulmonary congestion and dyspnœa.

As regards myocarditis from rheumatic fever, it usually attacks hearts already the seat of acute or chronic endocarditis, or is associated with pericarditis, although the muscle alone may sometimes be affected. For this reason it is not easy to recognise or definitely determine the myocarditic complication. Moreover, experience proves the folly of attributing to myocardial inflammation the cardiac asthenia, or even dilatation, so often witnessed in acute rheumatism, for it is often but a manifestation of the poison upon the function of the organ. At all events, it is the part of wisdom in such cases to refrain from a positive opinion.

In typhoid and scarlet fever it is not uncommon to observe during the height of the attack symptoms of heart weakness, which in most instances subside with convalescence, and which are perhaps the result of the action of the infection on the vasomotor centre in the cord, together with exhaustion of the heart-muscle. Nevertheless, one should guard against an inclination to look on all such manifestations as not due to myocarditis. The onset of acute inflammation of the heart-wall is often so insidious during the febrile stage, and the symptoms are so latent, that the real nature of the heart disorder is readily overlooked.

On the contrary, when after subsidence of all active symptoms referable to the primary disease and during convalescence the pulse begins to assume the characters already described—i. e., feebleness, emptiness, irregularity, and conspicuous instability—one should at once suspect the existence of myocarditis.

We frequently encounter individuals who have successfully weathered a severe typhoid fever many months, even two or three years before, and still display undue rapidity and even irritability of the heart's action. I am always inclined to speculate on the possibility of such patients having suffered from unsuspected myocarditis of mild form, and yet sufficient to have left its traces behind. Should the heart-muscle become inflamed during or after the subsidence of acute rheumatic manifestations, the symptoms of circulatory embarrassment will be out of proportion to the clinical evidence of cardiac disease, and yet are very likely to be attributed to such structural alterations as are discovered. There is far more feebleness, emptiness, inequality, perhaps intermittence, and particularly instability of the pulse, than there is evidence of visceral engorgement and mechanical obstruction in the extremities, at all events until sufficient time has elapsed for the endocardial mischief to become intensified by the myocarditis.

The symptoms of purulent myocarditis depend somewhat upon the nature and extent of the changes induced, but are essentially the same as in malignant endocarditis—i. e., rigors, intermittent fever, sweatings, and splenic enlargement. If an abscess of the myocardium breaks into the blood-stream, there are infarcts in the skin, kidneys, brain, or other organs, or in event of rupture of the heart-wall, collapse and death. The clinical picture is usually such as to direct attention to the heart as the seat of the disorder. Yet in cases of diffuse myocarditis like that narrated by Freund, symptoms referable to the heart may be few and obscure, wholly out of proportion to the gravity of the malady.

Physical Signs.—*Inspection.*—Pallor is said to be conspicuous, and, associated with either apathy or restlessness, is very suggestive.

Palpation.—The pulse is weak, empty, and strikingly unstable, and the cardiac impulse is feeble or absent. In some instances the liver and spleen may be palpable.

Percussion.—This may or may not disclose an increase in the area of deep-seated cardiac dulness, but if such increase is associated with the characters of the pulse just mentioned, it greatly strengthens the diagnosis.

Auscultation.—As a general thing the ear detects no more than enfeeblement and perhaps muffling of the heart-sounds. Murmurs are present only when dilatation leads to relative valvular incompetence or endocarditis or pericarditis is associated.

Diagnosis.—Unfortunately the diagnosis of acute myocarditis is usually a matter of conjecture, and reliance must be placed on the history of some infection that may act as an etiological factor, even more than on the symptoms and physical findings. During the height of an acute infection, as diphtheria, acute rheumarthritis, and typhoid fever, it may be utterly impossible to diagnosticate with certainty the existence of acute myocarditis, whereas the occurrence of the physical signs described during convalescence renders the presence of the disease highly probable. If in a case of suspected myocarditis phenomena of sepsis are observed, they point strongly to a suppurative process.

Prognosis.—It goes without saying that the prognosis is always grave, even in simple myocarditis, and in the purulent form is absolutely unfavourable. Although there is post-mortem evidence that small, scattered abscesses in the heart-wall sometimes undergo a process of cure, still a case that is sufficiently outspoken to be clinically recognisable is from its nature incurable. In acute interstitial myocarditis of diphtheria Romberg computes the fatal termination as taking place in about one-third of the recognised cases.

It is not unlikely that in rheumatism the percentage of recovery is larger. This would appear reasonable when we consider that the parenchymatous degeneration is not so intense as in diphtheria. Although all cases do not die, yet the possibility of sudden death should never be forgotten; and, moreover, this possibility is not wanting in any given case simply because the evidence of cardiac mischief is slight. It is often precisely in this class of cases that danger is most imminent, since the physician, patient, and friends are likely to be thrown off their guard, and hence permit or commit indiscreet effort.

When, on the other hand, symptoms of collapse appear, the danger of death is very imminent. Increasing acceleration of the pulse and marked instability, the heart evincing a degree of feebleness out of all proportion to the demand made upon it, are signs of great danger. So also is abnormal retardation of the pulse-rate, as is exceptionally observed. Delirium renders the prognosis more grave; and the occurrence of emboli is an exceedingly bad omen, since we cannot predict how many are likely to follow or where they will lodge. Favourable indications are to be found in a gradual return of strength, volume, and regularity to the pulse.

Treatment.—We possess no means of directly influencing the inflammatory process after it has attacked the myocardium, and therefore our efforts must, in the first place, be directed to the prevention of myocarditis, if possible, and secondly, to protecting the heart-wall from all extraneous influences which can intensify the damage it is sustaining from the inflammation. To the former end is the early employment of such measures as may lessen the activity of the primary disease, which in diphtheria involves the earliest possible use of the antitoxine. The harm that may result from delay in the employment of this remedy becomes at once apparent when we reflect on Romberg's statement that the symptoms of acute myocarditis may arise at a time subsequent to the administration of diphtheria antitoxine, which goes to prove that the longer the infection is at work in the system the greater the likelihood of the heart-muscle becoming affected. In streptococcus infection the antistreptococcic serum would certainly be indicated; but in scarlatina, typhoid fever, and rheumatism we have no specific remedy, unless with the exception of salicylic acid in rheumatic fever; and hence we must endeavour to promote elimination through the kidneys by administering copious and frequent draughts of water, and subcutaneous and rectal injections of physiological salt solution.

So soon as symptoms of myocarditis are detected the indication is to maintain the patient in absolute repose of mind and body. Physical effort is dangerous, and so long as cardiac weakness exists the patient must remain in bed. He should receive as much highly nutritious and simple food as he can assimilate—milk, eggs, broths, etc. The bowels are to be kept active, though depleting purgatives are to be avoided. Strychnine is highly serviceable, and should alcoholic stimulants or ammonia be thought indicated, they are to be administered.

The character of the pulse would appear to call for digitalis, strophanthus, etc., but if prescribed, it should be cautiously and tentatively, for we are not in position to predicate how much of the myocardium is left uninjured and capable of responding, or whether damage may not accrue to fibres that have undergone extensive degeneration. Præcordial pain and restlessness are to be allayed, and for this purpose there is nothing better than morphine.

In conclusion, it may be repeated that the agencies of greatest service are rest, food, strychnine, and stimulants, in the order mentioned. In diphtheria it is often perilous to allow the patient to even rise up in bed to take a drink or to evacuate bladder or rectum; he must be kept as motionless as possible. Moreover, it will often be necessary to retain the patient in bed for many weeks or months. A rigid enforcement of this rule, even though it seems hard and cruel, is in fact a display of greatest kindness. When at length such a measure of improvement has been reached that absolute rest is no longer needful, the patient is to resume exercise by degrees, and at first with the utmost caution. Under no circumstances is an attempt to ascend stairs to be permitted before weeks perhaps of gentle moving about the bed-chamber have proved that the heart is no longer unduly taxed by such efforts. During this period of convalescence cautious attempts may be made to strengthen the heart by resistance exercises and saline baths. At this time benefit may be derived from iron, arsenic, and other hæmatics and nerve tonics. It is needless to add that in the case of young children it is often most difficult, yet no less important, to enforce the quiet and other measures necessary.

CHAPTER XX

CHRONIC MYOCARDITIS

SYN.: *FATTY DEGENERATION—FIBROID DEGENERATION—MYOFIBROSIS—WEAKENED HEART—CHRONIC CARDIAC INADEQUACY*

By far the largest number of persons who at or after middle age begin to manifest signs of cardio-vascular disturbance are not victims of valvular disease. Clinically they present evidence of failing circulation with enlargement of the heart (hypertrophy with dilatation), and with more or less thickening of the arteries. In some instances certain symptoms, as angina pectoris, point unmistakably to coronary sclerosis, with its consequent alteration of myocardial nutrition, but for the most part there is nothing that serves as a criterion of the nature and extent of the change in the heart-muscle. The microscope has revealed not only a considerable variety of pathological changes in these cases, but also a want of uniformity or constancy in the relation of these to the symptoms. In other words, various myocardial alterations seem capable of producing the same clinical picture, and conversely, various clinical pictures appear to result from one and the same pathological change. There is consequently much confusion and uncertainty still regarding the pathogenesis and precise relationship of the pathological findings, so that in dealing with this phase of cardiac disease one is at a loss whether to attempt to consider it from the standpoint of the pathologist or of the clinician. In either case one is pretty sure to get himself into trouble. German writers, as Romberg and Rosenbach, group the cases under the head of Chronic Cardiac Insufficiency. The latter maintains that as the various changes in the heart-muscle are but different manifestations of one process, it is impossible to diagnosticate anything more than heart-weakness, while Romberg classifies the cases according to their etiological factors. Thus he considers in one group those due to coronary disease, those to obesity, those to strain, and those to nephritis, those to excessive consumption of beer, etc. Such a division is in accord with the uncertainty of our knowledge on many points, and also has the merit of simplicity, but it is open to the objection that we cannot always be sure of the exact etiology of all cases or of the precise mode of operation of supposed causes. It also, as he himself admits, necessitates much repetition, and therefore I have decided to deal with these cases under the heading given to this chapter. I am well aware of the objections to such a grouping, and know that many times it seems simpler, and non-committal in a sense, to diagnose them according to the gross clinical findings of hypertrophy or dilatation or idiopathic enlargement of the heart. Still in most cases the microscope shows more or less myocardial degeneration, and therefore I prefer the term Chronic Myocarditis.

Morbid Anatomy.—Under chronic myocarditis the anatomical conditions usually considered are those of fibrosis and fatty degeneration. Conditions interfering with the nutrition of the heart may produce either or both of these changes, or the fibroid may be dependent on the fatty change.

Fibroid degeneration of the heart-muscle, or chronic interstitial myocarditis, may represent the final or reparative stage of the various acute forms of the disease. It is then to be regarded as a conservative rather than as a pathologic process. Thus in the case of infarction of the heart, or *myomalacia cordis,* the necrotic area is invaded by young connective-tissue elements, which finally are metamorphosed into a firm fibroid cicatrix. Such areas of fibrosis, or scleroses, are large or small according to the size of the original lesion. Very large areas are rarely found, as the acute disease would have probably proved fatal. Except by the formation of other infarcts this process does not tend to progress. It is only when the occlusion of many small arteries has produced multiple scleroses that the function of the heart is impaired.

Extreme fatty or parenchymatous degeneration, leading to destruction of the muscle tissue, may be the cause of a progressive fibrosis of the myocardium. Often the destructive process preceding the interstitial increase is a coagulation necrosis. As a rule this process is not strictly diffuse, and the appearance on section is of numerous scattered streaks and spots of a grayish or whitish colour, which project slightly above the plane of the section. Interstitial increase is not always dependent on antecedent degeneration, but progressive atrophy of the muscle and increase of the fibrous interstitial tissue may occur *pari passu* in such a way as to render it difficult to determine which is the primary and which the secondary process. In this case the fibrosis is more evenly distributed than in either of the above cases.

Scleroses are most frequently observed in the wall of the left ventricle, near the apex, and on the posterior side in the upper two thirds, near the auricle, in the papillary muscles of the left side, and in the interventricular sæptum. The fibrous increase may cause a thickening of the wall of the heart—connective-tissue hypertrophy—or the presence of the connective-tissue elements may so reduce the tone of the wall as to cause it to yield to the intracardiac pressure with the formation of bulgings, the so-called partial cardiac aneurysm, or in extreme cases with rupture of the heart.

Fatty degeneration is manifested by a general paleness with streaks and patches of a yellowish-brown colour. The markings of such a heart have been compared to those of a faded leaf. The muscle is softer than normal and easily torn. Fatty degeneration is most common in the wall of the left ventricle near the apex, next in the right ventricle, the interventricular sæptum, and the right and left auricles in the order given. It usually affects the muscle close beneath the endocardium more than that near the pericardium, and the brownish or yellowish mottling is sometimes plainly observable from within the heart. Microscopically the protoplasm of the fibres is seen to be replaced to a greater or less extent by fat droplets. These are arranged in rows, and are said to be situated at the junction of the cross and longitudinal striations. Very advanced fatty degeneration leads to a disintegration of the fibres, and their consequent replacement by fibrous tissue.

Fatty overgrowth consists in an increase of the normal subepicardial layer of adipose tissue. This is normally noticeable only along the course of the large vessels, but in well-marked cases of fatty overgrowth the fat covers the entire organ and no muscle is to be seen. A thick blanket of fat over the heart acts as an effectual impediment to its work, but it is in the nature of an outside force, and not, as is the case in fatty degeneration, a disease of the muscle itself. Sometimes, however, the adipose tissue invades the subjacent myocardium, first penetrating between the fibres and later causing the complete atrophy and disappearance of the muscle elements. This process may penetrate the entire thickness of a ventricular wall, and of course greatly impairs its functionating power.

The senile heart presents a varying picture made up of elements from all of these conditions. As a rule the failing nutrition of old age induces a mixture of fibrous and fatty degeneration. Fatty overgrowth is common in those elderly persons who incline to obesity. The senile heart may be hypertrophied—this when associated with chronic nephritis and general arteriosclerosis—or atrophied in consequence of malnutrition and inaction. Very frequently this atrophy is combined with the condition of autochthonous pigmentation already described. This condition of brown atrophy is almost characteristic of the senile heart.

The nutritional disturbance which is accountable for these degenerations is frequently the result of the gradual narrowing or occlusion of the coronary arteries or their branches. This may be due to a sclerosis that is part of a general disease of all the arteries, or it may be due to obliterating endarteritis or to a local atheroma of the coronaries either at their orifices or branches. This local process is more apt to take place in the descending branch of the left coronary artery, and this accounts for the special predisposition of the apical portion of the left ventricular wall to fatty and fibroid degeneration.

Thrombosis or embolism of the coronaries induces the condition of *myomalacia cordis* already considered. The walls of the arteries may become of bony hardness, or atheromatous in patches. The terminal branches may be converted into fibrous cords impervious to the circulating fluid. If the obliteration of one artery takes place gradually the circulation may be established through branches of the other. The reduction of the blood-supply to the parts affected, and especially the lack of oxygen, induces fatty degeneration, and often subsequent fibrosis. The heart-muscle, probably on account of its constant activity, feels immediately any lack of oxygen, and hence the myocardium is especially prone to fatty degeneration.

Changes in the myocardium are almost always found associated with the various valvular lesions, and with hypertrophy and dilatation of the heart from any cause.

Etiology.—The changes of the heart-muscle which I have chosen to group under the generic term of chronic myocarditis are of slow development, and presuppose the protracted working of influences injurious to the function of the organ. These influences are for the most part conditions which cause a disproportion between the demands made on the heart and its nutritive supply—in other words, which require the heart to work in excess of its nutrition. The influences which put an abnormal demand on the heart may reside within the organism or may come from without, or there may be a union of both.

Under the first head are degenerative changes of the vascular coats and chronic kidney diseases, conditions which persistently augment peripheral resistance. Conditions residing outside the body are those which produce long-continued overstrain, as manual toil, the hardships of the soldier's or the sailor's life, the toilsome daily exertions of the mountaineer, excessive consumption of beer, etc. In many instances influences from within and without are conjoined. Something more is required, however, than mere increase of work, and this is to be found in disorders of cardiac nutrition. The blood itself may be of poor quality, or it may be vitiated by toxines of one kind or another, or the blood remaining healthy, its supply to the heart may be curtailed. It is in the first way that fatty degeneration of the heart is brought about by wasting diseases, cancer, chronic suppurations, repeated loss of blood, secondary and pernicious anæmia, exhausting discharges, as chronic diarrhœa, by insufficient food, etc. The blood may be vitiated by the toxines of acute infectious diseases, by chemical poisons, and probably by toxic substances developed within the gastro-intestinal tract, some of them of bacterial and some of putrefactive origin. Typhoid and scarlet fever, diphtheria, acute rheumatism, and influenza are all capable of setting up not only acute myocarditis, but chronic myocardial changes of an allied if not identical, yet of a more slowly acting nature. Phosphorus and arsenical poisoning are well-recognised causes of fatty degeneration of the heart.

The myocardial degeneration of chronic kidney disease may be due in part, at least, to chronic toxæmia acting in conjunction with prolonged high arterial tension. The degenerations depending upon coronary sclerosis are instances of the third kind—i. e., of defective blood-supply.

When the two great factors, work and excessive or deficient nutrition out of proportion to that required for the work, are combined, then we not only have myocardial degeneration, but in time also inevitable cardiac inadequacy.

The hypertrophy which so often develops in association with chronic myocarditis is the expression of an attempt at compensation. It probably evinces an effort on the part of Nature to repair the damages going on in the heart, but it also results from the necessity on the part of this organ to overcome peripheral resistance.

Fraentzel's idiopathic enlargement of the heart was thought by him to result from the consumption of an amount of food in excess of the requirements of the organism and of the individual's bodily exercise. Hence it is found in its most typical form in persons who are large eaters, and who, in consequence of their particular line of work, are compelled to be sedentary. According to Rosenbach, physical inactivity is an important element in this class of cases. When these individuals reach middle age they are usually found to have developed corpulent abdomens, and they generally continue to increase in weight. In many at this time the previously existing high-pulse tension is still further augmented by degeneration of the blood-vessels and kidneys, often also of the liver, which retrograde changes are probably to be referred to the same etiological factors. So long as cardiac hypertrophy enables the organ to meet its demands its functional integrity is intact. At length, however, either because its nutrition has suffered to such an extent that it cannot meet the ordinary demands made upon it, or because extraordinary work is suddenly required, as from some undue physical effort, the heart finds itself overpowered, and symptoms of myocardial incompetence set in.

In the working classes, in soldiers, in sailors, and mountaineers, in persons addicted to the abuse of beer and other alcoholic beverages, who at the same time perform manual toil, influences of various kinds are active. Food defective in quality or quantity, privations and hardships, and toxic agencies serve to intensify the injurious effects of overstrain. In southern Germany, notably Munich, hypertrophy and degeneration of the heart in its most typical form are attributed to the excessive consumption of beer. Some have thought this due to the great vascular strain incident to the daily intake of many litres of fluid, but Krehl, Rosenbach, and others recognise in addition the etiological influence of toil and the nutritive elements contained in the beer as well as of the strain put upon the circulatory apparatus by the consumption of excessive amounts of the fluid. In soldiers and mountaineers who carry heavy knapsacks strapped upon their shoulders, and thus loaded perform wearisome marches day after day, Rosenbach thinks cardiac function is impaired through respiratory embarrassment occasioned by constriction of the chest, and through the necessity of overcoming abnormal peripheral resistance. Athletic sports, as well as coffee, tobacco, and alcohol, he considers injurious only in the abuse, not their use.

Myocardial degeneration from coronary sclerosis is an expression of inadequate blood-supply, either circumscribed or general; and that one may understand the diverse appearances encountered I think it well to quote in a general way Leyden's views of the mode of production of the changes in this class of cases.

He divides these into four groups in accordance with the degree of coronary changes and the rapidity with which blood-supply to the heart-muscle is shut off, as follows:

(1) The coronary arteries present more or less evidence of sclerosis, but are still able to supply the heart with sufficient blood to maintain its nutrition. Degeneration does not take place, and the organ performs its functions without symptoms referable to coronary disease. Death results from an intercurrent affection, and knowledge of any alteration of the coronary vessels is but the accidental revelation of an autopsy.

(2) One of the coronary arteries, usually the anterior descending branch of the left, which has become thickened, subsequently undergoes obstruction by thrombosis. When this takes place slowly, or when the circulation is but imperfectly cut off from the area supplied by the affected vessel, the wall of the heart within this space undergoes fatty or fibroid degeneration. If the thrombosis, on the other hand, suddenly and completely deprives

the part of its nutrition, then this area breaks down into the "*myomalacia cordis*" of Ziegler. Occasionally the extravasation of blood into this softened area gives it an appearance of a hæmorrhagic infarct. When rupture of the heart occurs, it is generally within such a spot of acute softening.

(3) Sclerosis of the coronary arteries is general and has come on gradually, giving rise to correspondingly gradual changes in the heart—i. e., fibroid degeneration either circumscribed or general. When this chronic or fibrous myocarditis is diffused, the ventricular walls are apt to be thin and dilated, whereas circumscribed areas of induration are frequently associated with hypertrophy. In rare instances this development of new connective tissue is attended with atrophy of the muscle-fibres, and the organ shrinks in size after the fashion of a cirrhotic kidney.

(4) In this group, the coronary sclerosis, although essentially chronic, has been hastened and intensified from time to time by acute exacerbations of the process, thrombosis, etc. The changes in the heart are therefore twofold; areas of acute softening and fatty degeneration are interspersed among those of chronic myocarditis. This group, which blends the second and third, therefore, is the one most often encountered by the physician.

The causes of coronary sclerosis are obscure, but are probably those of arteriosclerosis in general. That age is of influence seems attested by the fact that more or less evidence of the change is found post mortem in persons past middle age, while it is rare under forty and wanting in children. Some families seem to present a remarkable tendency to sclerosis of the coronaries and consequent myocardial disease. This has led to the suggestion of a possible hereditary influence in its production. Some individuals appear to be endowed with "arterial tissue or vital rubber of poor quality, which cannot be explained in any other way," as is so aptly expressed by Osler, "than that in the make-up of the machine bad material was used for the tubing." In my experience individuals who display this tendency to early sclerosis usually manifest distinct gouty diathesis. They may be said to be suffering for the sins of their ancestors. I have long had the conviction that in some families the heart is the *locus minoris resistentiæ*, in some displaying particular vulnerability to the rheumatic poison, and in others appearing unable to withstand the wear and tear of modern business life. Certainly it is not very uncommon to elicit from a patient, himself suffering with myocardial disease, a history of a parent, usually his father, and one or more of his brothers and sisters having died suddenly of heart-disease.

I have in mind now a family in which the father is reported to have died of "ossification of the heart," while of three of the seven sons one had attacks of angina pectoris, another had heart-disease, developed at middle age, and the third died suddenly with a dilated heart, after having suffered from one or two outspoken anginal paroxysms.

Males suffer from chronic myocarditis more often than do females, but this is probably owing to their greater exposure to those conditions favouring the development of myocardial incompetence rather than to any inherent tendency residing in the fact of sex alone. Women frequently manifest clinical and post-mortem evidence of cardiac degeneration, and according to Rosenbach, it is particularly those who bear children in rapid succession, and are still further depleted by lactation and insufficient nourishment.

The immediate causes of cardiac incompetence cannot always be ascertained. It not infrequently develops as a direct result of heart-strain through indiscreet physical efforts; but it also may appear without any such determining factor, and is then the end-act of all those factors that have led to the degeneration. Worry, grief, excessive business cares, as in times of financial stress, and even emotional excitement of other kinds, may not only occasion loss of power in hearts already the seat of myocardial disease, but are said to be an etiological element in the development of the muscle-disease itself.

The cardiac inadequacy of chronic nephritis is generally the result of the organ's inability to longer withstand the excessive tension in the vascular system. It may develop slowly or may be precipitated by some extra exertion or other source of added heart-strain.

Symptoms.—The cardiac incompetence of myocardial disease displays clinical pictures of considerable variety in detail, yet which possess the same fundamental characters. It may be seen as Fraentzel's Idiopathic Enlargement of the Heart, as the Senile Heart so graphically portrayed by Balfour, as a case of angina pectoris from coronary sclerosis in nocturnal attacks of dyspnœa, known as cardiac asthma, and in connection with chronic nephritis or diabetes, or both, and occasionally as a mitral or aortic regurgitation due to relative insufficiency from dilatation.

Idiopathic cardiac enlargement occurs for the most part in middle-aged men of powerful physique, who are intellectually active, but physically inactive. It is especially frequent, therefore, in men of affairs, as merchants and railroad magnates, and in professional men, as lawyers and clergymen, who, in addition to sitting for long hours at their desks, generally consume large amounts of food. For years there is in such individuals an abnormally high and sustained pulse-tension, which resulting, perhaps in part, from abnormal blood-pressure within the abdominal cavity, increases as the girth of the waist increases. This state of things is borne without special discomfort until the man gets well along in his forties, or has even passed his fiftieth year. Then he begins little by little to notice he does not breathe quite so easily as formerly on ascending stairs, walking up a slight hill, or hurrying to catch a street-car. At times, particularly after breakfast or a more than ordinarily hearty meal, he finds that walking at his accustomed pace is attended by a feeling of uneasiness, fulness, or even dull pain in the region of the heart. As the weeks go on he finds these two symptoms become decidedly annoying, and instead of wholly subsiding after he sits down they remain as a vague sense of discomfort in the chest. He also perceives, perhaps, that the old exertion or some excitement incident to his occupation produces consciousness of his heart's action, a veritable though not violent palpitation. As a rule this last symptom is not at all pronounced, being subordinate to the breathlessness and præcordial fulness.

By the time things have reached this pass he concludes to consult his physician, who finds a strong, usually regular pulse, no cardiac impulse, an apparently normal heart's dulness, and clear heart-tones without murmur, but the aortic second sound decidedly accentuated. If the radial arteries are not noticeably stiff, and the urine is negative, the real nature of the case is apt to be overlooked, and the symptoms are attributed to the man's increasing weight. He is told to exercise more, eat rather less, and not to worry. Perhaps he is advised to go to some springs, where he can drink laxative waters, or he goes thither on the recommendation of some friend. At first the waters and active exercise seem to make him feel better, but after returning home, and having resumed his former mode of life, his symptoms reassert themselves, this time in greater intensity. He again seeks his physician, who now finds the pulse is accelerated, the heart a little enlarged, and the liver palpable. The urine is scantier than before, and it maybe contains a trace of albumin. He is put upon digitalis and strychnine, or he is sent to Bad Nauheim. This treatment improves his condition to a greater or less degree. His symptoms are lessened or disappear entirely for a number of months. Then, in consequence of return to his old ways and his neglect of his doctor's injunctions, he finds his enemy has again attacked him. The former course of treatments is repeated with less brilliant results. He is now permanently crippled, but is still able to attend to a part of his duties. As months go on, however, his shortness of breath and other symptoms of cardiac inadequacy grow more and more pronounced, therapeutic measures are less and less effective, and at length this once powerful and active man of affairs is laid by, a pronounced sufferer from dyspnœa, hepatic stasis, increasing œdema, scanty, perhaps albuminous urine, insomnia, a dull, congestive headache, cough, and frothy, or even blood-tinged sputum, a feeble and often arrhythmic pulse, a dilated and feeble heart—in short, all the signs of advanced cardiac insufficiency.

In other cases the course of the disease from initial breathlessness to complete breakdown of heart-power is much more rapid. Instead of extending over two, three, or more years, it passes through its several phases in a few months, or even five or six weeks, as I have more than once observed. In some instances the clinical history is merely that of ever-increasing cardiac debility, while in others there are some of the special features, as cardiac asthma, bradycardia, or even the so-called Stokes-Adams symptoms, which are described in full in a special article. Whatever the variety of colouring, the general picture is that of more or less rapidly progressing loss of heart-power, and the ultimate outcome is always the same.

Cases of chronic myocarditis are conveniently divided into three great groups, according to the predominance of their clinical manifestations. These are:

(1) The arrhythmic form, in which the pulse is strikingly irregular and intermittent, now slow and strong for a few beats, now perhaps rapid and feeble, or again made up of a perfect jumble of large and small, distinct and imperceptible, slow, rapid, intermittent waves that seem to fairly tumble over each other in their hurry, or to lag back until driven hastily onward again by the impetuously rushing waves behind. In a word, the pulse is so lacking in regularity of rhythm, force, and volume that to count it accurately is impossible.

(2) This is the form characterized by tachycardia and called the tachycardial form. The pulse is persistently accelerated, or in a few instances become so, in paroxysms which so annoy or even terrify the patient that he comes to stand in mortal dread of his attacks of palpitation.

(3) The *asthmatic* form, distinguished by attacks of acute pulmonary œdema, which not only occasion distress and terror to the patient, but throw the friends into a state of scarcely less alarm. I have under observation at the present writing a powerfully built, active business man of sixty-three, with moderately stiff arteries and a hypertrophied heart, with feeble tones, and a scratching systolic murmur, who for the past several months has lived in a state of well-nigh intolerable nervousness and apprehension. As he says, he has completely lost his nerve, because last September, after some weeks of neglected shortness of breath, he was one evening seized with an attack of urgent dyspnœa, during which the pulse was scarcely perceptible, and the chest emitted a multitude of fine crackling sounds. He coughed from time to time and expectorated a frothy white sputum. The attack subsided after the hypodermic administration of morphine and atropine. This gentleman has had one recurrence of the kind, but in the meantime has scarcely passed a week without hours or even days during which his heart has "thumped and bumped," to use his expressive words, in a manner which throws him into a state of great alarm. He never knows when this palpitation is to occur, but it seems in some way connected with temporary augmentation of pulse tension. It is also quite certain to follow excitement over business affairs.

The pulse-rate averages from 88 to 95, but often runs up to 120 or even 140, and such is the throbbing that he can at any time count his heart-beats without feeling his radial pulse. There is danger of death during his attacks and he knows it, which of course keeps him in a state of hourly apprehension. The foregoing case shows the occasional blending of the tachycardial and asthmatic types.

Patients of the arrhythmic form are very common and display the greatest tolerance of the really serious cardiac condition. Thus I knew a man of fifty-five who, notwithstanding an enormously dilated heart and exceedingly arrhythmic pulse, managed to drag on for nine years from the beginning of symptoms. These were not very severe; panting respiration on exercise, a feeling of weakness, so that he could not attend to business, and considerable fermentative indigestion. That was about all, and at last his end came through ascites rather than distinctively cardiac inadequacy.

It must not be supposed that cases of chronic myocarditis can always be clearly separated into the forms just described. Many of them blend the symptoms belonging to each in a way to make a very complex clinical picture. Neither are the disturbances of which these patients complain always strictly cardiac. These latter may be said to be dyspnœa, heart-pain, palpitation, visceral congestions, attacks of pulmonary œdema, cough, and dropsy. They are all present in varying proportion, not perhaps in every case, but in many cases. In addition, however, there are very sure to be numerous other complaints which in all likelihood depend more or less remotely upon the disordered circulation and perverted visceral function arising from disordered blood-flow. Vertigo, insomnia, neuralgias and myalgias, nervousness, irritability of temper, indefinite sensations in the region of the heart, numbness, and formication—in short, a score of sensations which the patient connects directly with his heart, and calls on the medical attendant to explain and relieve. They are a large part of the daily plaint of these chronic sufferers when able to be about and in a state of partial compensation. When, however, really serious symptoms of cardiac insufficiency set in, they are so much worse that they drive away more trifling sensations and dominate the scene.

All cases do not fall distinctly into the class of Idiopathic Cardiac Enlargement or of the Senile Heart, but occupy a sort of intermediate ground. Nevertheless, it is conducive to clearness to try to classify them, as is essayed to do in this chapter.

The senile heart forms but a part of a general degenerative process. In one case the arteries are markedly stiff and tortuous; in another the urine shows evidence of pronounced interstitial nephritis, but in all the phenomena of cardiac incompetence dominate the scene. There are breathlessness on even slight exertion, feebleness, digestive disorders, sensitiveness to cold and changes of weather, a tendency to bronchitis, and insomnia. In some there are attacks of nocturnal dyspnœa of greater or less intensity, flutterings of the heart, vertigo, or even syncopal attacks.

In other cases the breathing assumes more or less typically the Cheyne-Stokes type (see article on Cheyne-Stokes Respiration), with or without œdema, pulmonary congestions, arrhythmic feeble pulse, and the usual manifestations of progressing asystolism.

The course is usually slow, the symptoms being sometimes mild, and the patient cut off by some intercurrent affection, as senile pneumonia. In some instances there is history of attacks of angina pectoris for five, ten, or even twenty years, and death at last is sudden and unexpected. There are other cases, chiefly men, whose cardiac inadequacy is shown by acceleration and arrhythmia of the heart's action, inclination to cough and wheeze, and various so-called gouty manifestations rather than by pronounced dyspnœa or venous stasis; the so-called arrhythmic form. They get rather breathless on exercise, and yet then can walk at a moderate rate of speed without much difficulty. They have times when from some illness, as acute bronchitis, injudicious strain of one kind or another, they are laid up in their room with a trace of œdema and indications of failing heart-power that look very threatening, and yet under good nursing and proper medical attention they rally, and after weeks or months are again able to be about, a little weaker and thinner, a little more breathless, but on the whole capable of getting considerable enjoyment out of their quiet existence.

I recall an old gentleman of eighty with stiff arteries, urine of poor quality, and a greatly hypertrophied heart, the first sound accompanied by a loud systolic basic murmur, the aortic second intensely ringing, who yet attended daily to the cares of a large personal property besides many other duties of a public and private nature. At length one spring he and his friends noticed that he began to breathe with difficulty upon ascending stairs, and at rest displayed a peculiar sort of breathing which they had never observed before. Notwithstanding this difficulty he came to consult me at my office, and seemed surprised when he was told to return home, give up his business, and remain in the house. His pulse was rather unstable, occasionally intermittent, but not notably accelerated. His respiration was only moderately dyspnœic as he moved about the room, but after he had been sitting still for some time, and particularly when asleep, it became irregular, with short periods of nearly but not quite complete cessation of breathing, which were then succeeded by gradually deepening inspirations until they grew full and vigorous. They then died away rapidly into apparent apnœa. Close observation detected that at some of these times he yet breathed faintly, while at others he breathed not at all. Some of the dyspnœic periods were longer and more pronounced than others, and for minutes together others of them presented all the characters of typical Cheyne-Stokes respiration.

During these periods he did not seem subjectively conscious of distress. Under appropriate treatment of a stimulating and eliminating kind and prolonged confinement to one floor, afterward to the house, his irregular type of breathing gradually left, the heart grew steadier and stronger in action, and he came to look upon himself as pretty well. He required rather close watching to prevent indiscretions, chiefly in way of exercise, but as time went on he was able to transact a little business and to enjoy the visits of his friends. In this manner this gentleman was able to live on for two years. When at length the final struggle came, it was in the form of a renewal not of incomplete Cheyne-Stokes respiration, but of a most distressing dyspnœa, which gave him no peace even when quiet in bed. It was not attended with notable signs of cardiac failure, and no particular evidence of renal insufficiency. It may have been due to bulbar sclerosis; but at all events it at length necessitated the hypodermic administration of such heroic and frequent doses of morphine, that he at last expired in a state of complete narcosis.

In the case of a man of seventy-one, who had been a well-known journalist, the initial symptom so far as could be ascertained was an attack of dyspnœa, which seized him one night after he had retired. He fell asleep, and after a few minutes sprang up in bed, clutching at his throat, exclaiming he was going to strangle. This so terrified him that at length he arose, dressed, and compelled his valet to keep him walking up and down in the garden for the remainder of the night. When I saw him the following afternoon he was still greatly agitated and suffering from choking spells. A hypodermic of an $\frac{1}{8}$ of morphine, with atropine in the ordinary combination, gave six hours' uninterrupted sleep the next night, although during his repose his breathing was typically Cheyne-Stokes. This patient's pulse was very arrhythmic, his arteries sclerotic, his heart dilated, with a blowing apex-murmur and feeble sounds. His urine was that of a moderate renal cirrhosis, and he suffered from prostatic enlargement. His stomach was dilated, and he had any amount of flatulent distention of the bowels. Altogether it was an unpromising case, yet persistent and vigorous treatment with cardiac tonics and cathartics, with as strict a control of the dietary as was possible with an irritable, self-willed old gentleman, at length pulled him out of his deplorable condition. For four years he was an invalid, having times of profound physical and mental depression, and periods of grave cardio-vascular disturbance, during which œdema more than once appeared. These periods of distress were alternated with seasons of comparative immunity from symptoms, and yet they were interspersed with numerous attacks of bronchitis, mild uræmic manifestations, and once an acute pleurisy with effusion that eventually necessitated paracentesis.

More than once he rallied from what it seemed must prove his final illness; and thus actually kept alive by cardiac tonics, he managed to drag out four years of chronic cardiac inadequacy. When at length his end came, it was quite sudden, although preceded by days of more than usual feebleness that had confined him to his bed. Although this patient displayed marked general debility, with slowly increasing cardiac asthenia, he never again suffered to any extent from his nocturnal dyspnœa, and never had an attack of angina pectoris. He frequently complained of obstinate chest pains, but these were unmistakably an intercostal neuralgia, as shown by numerous hyperæsthetic areas.

This case belonged to what may be styled the arrhythmic group, in which the cardiac insufficiency is shown by such an irregularity and intermittence of the heart's action that it constitutes a veritable *delirium cordis*.

Radizewsky has shown, conclusively as it seems to me, that when the pulse displays these characters there is extensive fibroid degeneration, chiefly of the auricles, which both clinically and post mortem are found dilated. In his communication upon the subject he quotes Hampeln's researches, which demonstrated that perfect regularity of the pulse is frequently seen in cases in which subsequent necropsy discloses extensive fatty degeneration of the left ventricle. In other words, the state of the ventricle cannot be determined by the state of the pulse. Radizewsky's findings have always seemed to me to explain the protracted course which so often characterizes cases in which the pulse is strikingly arrhythmic.

Degeneration and dilatation of the auricles impair the functional integrity of the heart-muscle, but can never so seriously threaten life as when the wall of the left ventricle is degenerated. I have seen many of these cases with unmistakable cardiac inadequacy drag along for months and even years after the heart had become so feeble and arrhythmic that it seemed impossible for it to maintain the circulation.

On the other hand, experience has taught me to dread those degenerated and senile hearts, which are apparently not much dilated, yet give rise to dyspnœa of effort, while the pulse remains accelerated, but perfectly regular. Such hearts are usually refractory to treatment, and are apt to surprise one disagreeably by stopping suddenly and unexpectedly. They belong to the tachycardial form of chronic myocarditis.

In my experience the clinical picture of the senile heart is very rarely that of great dropsy and extensive visceral congestion with overdistention of the cardiac chambers, as in the terminal stage of Fraentzel's Idiopathic Enlargement of the Heart.

In the *heart with coronary sclerosis* the consequent interference with nutrition of the heart-muscle leads to changes of a chronic nature in the majority of cases. In others, circulation within the coronary arteries is more or less suddenly shut off, and the heart-muscle suffers with corresponding acuteness.

Consequently the symptoms due to coronary disease are diverse, and are best divided, as by Leyden, into acute, subacute, and chronic.

In the acute form the clinical history is confined to a few days, hours, or even minutes. The symptoms are due to coronary thrombosis with secondary myocardial softening, to sudden rupture of the heart in some area of insidious fatty degeneration, or of acute molecular necrosis (myomalacia cordis), or the symptoms consist only of a sudden diastolic arrest of the heart's action.

The symptoms of *heart-rupture* may be a sudden sharp attack of angina pectoris or of vague præcordial distress and oppression with profound prostration. The action of the heart is feeble and disordered, and the organ fails to respond to stimulants. The intellect is generally clear, but unconsciousness may be present. In some cases there are symptoms of gastric disturbance, even vomiting, and the case is thought to be one of gastric disorder. If life is prolonged for several days, as very rarely happens, the first violence of the attack abates, not to be renewed, or the angina and præcordial distress recur from time to time with steadily increasing asystolism and death. In other cases of cardiac rupture the symptoms are chiefly those of rapidly failing circulation with insensibility and death from acute pulmonary œdema.

With the onset of symptoms the nearest physician is hastily summoned, and on arriving at the patient's side finds him moribund. Stimulants prove inert, and the doctor signs the death certificate without having been able to accurately diagnose the condition. If he examines the heart he discovers clear feeble sounds without any appreciable increase in the area of dulness. In other cases cardiac dulness is increased, but unless a slow escape of blood into the pericardium produces the outline characteristic of pericardial effusion, the augmentation of dulness is attributed to dilatation, and a diagnosis is made of paralysis of the heart (acute asystolism), which seems borne out by the weakness of the sounds and strikingly poor quality of the pulse. In such a case recently narrated to me death supervened in half an hour from supposed acute dilatation, and yet the sac was found absolutely distended with blood.

In another form of this acute type of coronary sclerosis the patient appears as well as ordinary, having made no complaint that led to a suspicion of his having heart-disease, and yet in the midst of his activities, while at work in his office, on rising from dinner, etc., he suddenly falls dead. He may turn pale, speak of vertigo, give a groan, or in some way attract the attention of his family, who spring to catch him as he sinks to the floor in fatal syncope. The mahogany flushing of the face and few gasping inspirations suggest death from apoplexy, but in reality it is from sudden diastolic arrest of the heart. In such instances the degenerative process has invaded some of the vital centres in the heart, probably in the upper portion of the interventricular sæptum.

In cases which, according to Leyden's classification, may be called subacute, there are attacks of angina pectoris extending over a period of weeks or months. They differ much in different cases, as regards severity and frequency of occurrence. As a rule, however, they grow more intense and more frequent. The sufferer speedily becomes incapacitated for business, keeps in the house, or ventures forth only on mild, still days, grows daily weaker and paler, and is very apt to lose flesh. Death comes at last either from slowly increasing asystolism, or suddenly during an anginal paroxysm. The pulse in such cases is usually regular, and but moderately if at all accelerated, while examination of the heart is generally negative. The arteries may display some stiffness, but aside from the patient's age and his attacks of pain there is little to indicate chronic myocarditis.

Cases coming under the chronic head run a slow course and extend over years, instead of months, five, ten, or even twenty. The individuals are always conscious of their liability to an attack, and hence deport themselves most circumspectly, eating and drinking moderately, walking and exercising carefully, avoiding raw cold winds, and shunning excitement and provocation to anger, lest at once their enemy be upon them. Their general health does not suffer greatly at first, although as years go by they look and act like invalids. A few experience some shortness of breath, or may exceptionally suffer from veritable cardiac asthma. As a rule, however, their one symptom is their terrible angina. Their pulse is generally regular, always appreciably tense, and their hearts are negative on examination. Death comes through intercurrent disease or during an attack. For further particulars the reader is referred to the chapter on Angina Pectoris.

In *general* arteriosclerosis there are often symptoms of circulatory failure which are apt to be attributed to cardiac inadequacy alone, when in reality it is the arterial stiffness that is responsible for the stasis and œdema. The heart may in such be atrophied or of normal size, but as a rule it is hypertrophied. The condition of the myocardium depends upon the degree of its nutrition with relation to its work, and as the aorta and coronaries share to a greater or less extent in the sclerotic process, the heart-muscle is not intact. Nevertheless, its driving power is often adequate for years after the blood-vessels have become rigid and beady. At length it reaches the limit of its compensatory ability, and vascular resistance still augmenting, the heart-walls begin gradually to yield to the strain of maintaining arterial circulation, and dilatation slowly supersedes hypertrophy.

In some cases injudicious physical effort, suddenly or too often exerted, causes acute overstrain of the already too greatly taxed heart, and symptoms of cardiac insufficiency set in rapidly instead of slowly. These are not peculiar, but possess the ordinary characters of retarded circulation, breathlessness, slight œdema of the ankles, scanty, perhaps albuminous urine, pulmonary and hepatic congestion. The superficial veins stand forth still more prominently, the rigid, tortuous, uneven arteries show a thready, often flickering pulse, which is accelerated and often irregular both in force and rhythm. The heart is found more or less increased in size, and its sounds are altered in quality and proper relative intensity. There may or may not be murmurs at apex or base indicative of atheromatous changes in the valves, but the aortic second tone is nearly always ringing and metallic.

The patient loses strength, and finally takes to the house permanently. Dyspnœa grows apace and not infrequently assumes the characters of cardiac asthma or of Cheyne-Stokes respiration. Urine grows still scantier, dropsy advances, orthopnœa sets in, troublesome cough, and frothy, blood-tinged expectoration, betoken ever-increasing stasis within the pulmonary vessels, and the patient succumbs after weeks or months to general exhaustion, cardiac asthenia, or an attack of acute pulmonary œdema.

Chronicity is the essential feature of this type of cardio-vascular inadequacy. It is not uncommon for cases of arteriosclerosis to drag on for several years under the picture of senility and general decrepitude, and death to come at last as a result of acute bronchitis, pneumonia, or even of renal inadequacy.

Exceptionally, although the total number of such cases is not small, the termination is through cerebral thrombosis. Rupture of a blood-vessel in the brain is not so frequent in senile arteriosclerosis as in younger persons whose renal cirrhosis leads to enormous left-ventricle hypertrophy.

Chronic nephritis leads to very serious changes in the heart-muscle. Clinically, this is shown by hypertrophy of the left ventricle either alone or as a part of a general cardiac enlargement. The myocardium may or may not be seriously degenerated, but whether it is or not, it is subjected to an enormous peripheral resistance to successfully cope with which it is compelled to undergo hypertrophy. I do not intend to discuss the various theories which have been advanced to explain this increase; for these the reader is referred to works dealing with kidney diseases.

The pulse of chronic nephritis shows prolonged high tension, which is primarily of renal, not cardiac, origin. If the kidney changes are of slow development, as in chronic interstitial nephritis, or if the patient is not carried off by the sudden onset of renal inadequacy, there surely comes a time when the heart, struggling with abnormally high endocardial blood-pressure, is able only with difficulty to withstand the enormous resistance in the arterial system. Then gradually or suddenly, according to circumstances, the wall of the left ventricle gives way.

If slowly, the volume of urine begins to fall off, and the patient finds his wonted physical efforts are attended by shortness of breath and palpitation, and his pulse-rate is found to be decidedly augmented. Signs of venous congestion and pronounced cardiac dilatation are usually wanting at this stage. Unless the danger is recognised and means are resorted to of restoring the equilibrium between pulse-tension and heart-power, cardiac insufficiency grows daily more apparent, and the patient is at length compelled to remain inactive. Examined at this time, he is found to evince unmistakable signs of failing circulation. The pulse is rapid and perhaps of poor quality; the liver is palpable and more or less tender; tension, and it may be pitting of the ankles, is detected; the tongue is coated, breath foul, and the urine is apt to be decidedly scanty and albuminous. If the heart is examined, the apex is displaced and lacking in concentration and force, the second sound at the right of the sternum is somewhat enfeebled, and the pulmonic is nearly or quite as intense.

The striking alteration in the heart-findings consists in the peculiar reduplication of the sounds at the apex, which is known as gallop rhythm (see introductory chapter). Occurring in the course of chronic Bright's disease this phenomenon is of very evil portent, for it indicates that the left ventricle is yielding to the abnormal strain and tottering on the verge of an irreparable breakdown. In most cases treatment is unavailing, symptoms of stasis progress, and dyspnœa becomes most distressing. It may be of the Cheyne-Stokes type, but is more often of a paroxysmal nature, coming in waves, as it were, with evidences of great agitation, even alarm, on the part of the patient, yet without corresponding signs of more than usual cardiac failure. This form of dyspnœa is probably partly of toxic (uræmic) and partly of cardiac origin. Nevertheless, the insufficiency of the heart augments, renal excretion fails correspondingly, and after the lapse of weeks or a few months the patient dies with uræmic manifestations or from acute pulmonary œdema.

There are other cases of Bright's disease in which cardiac incompetence sets in abruptly. This is usually owing to some indiscreet effort or excess which causes rapid dilatation of the left ventricle. The symptoms are much the same as in the more gradually evolved loss of compensation, but are apt to be of far greater intensity. Examination shows feeble apex-beat, displaced far to the left and perhaps downward, enormous increase of cardiac dulness, and a systolic apex-murmur, which often replaces the first sound, and a feeble second tone except in the pulmonary area, where it is intensified. The liver swells rapidly, is often painful and very tender, particularly in the epigastrium. Dropsy sets in, extends rapidly upward, and invades the serous cavities. The patient suffers from orthopnœa with paroxysmal exacerbations, from severe, tensive headache, insomnia, or it may be somnolence, and many other distressing symptoms of combined cardiac and renal inadequacy. Few clinical pictures are more distressing, and *none are more hopeless.*

In many cases in which there are enlarged hearts with stiffened arteries and urinary findings of renal sclerosis it is difficult to say whether the symptoms of failing circulation are due primarily to incompetence on the part of the heart or of the kidneys. Some of these patients manifest symptoms of slowly failing heart-power for many months before being compelled to regard themselves as hopeless invalids. I recall one gentleman of fifty-eight with this combination of cardio-vascular and renal degeneration, who, nearly two years before his death, suffered from paroxysms of dyspnœa which because of his rapid, unsteady pulse was thought cardiac, but seemed to me in reality uræmic. It did not yield until pulse-tension was reduced by frequent doses of nitroglycerin. Another gentleman of forty-seven with the same association of diseases used to complain that he could not breathe "more than an inch deep." This patient's heart manifested clinically the most enormous enlargement I have ever seen. His breakdown was initiated three years before by a "century run" on his bicycle.

Diabetes mellitus occurring after middle age, and usually conjoined with vascular and renal changes, is often seriously complicated by symptoms of cardiac incompetence. The arteries are more or less stiff, the heart is hypertrophied and dilated, and its action is rapid or pounding, sometimes intermittent. Glycosuria is the feature which has especially to be combated, and yet one must never lose sight of the cardio-vascular symptoms. At the present writing I have under observation two ladies who have diabetes mellitus with atheromatous arteries and hypertrophied hearts. In one, whose age is not far from seventy, the main complaint (so long as strict diet keeps down the glycosuria) is of great weakness, palpitation, and shortness of breath upon exertion. The other patient, of about sixty, suffers chiefly from dyspnœa, attacks of palpitation, and faintness. On two occasions in the early morning hours she has been awakened by a sense of suffocation, and has nearly died from acute pulmonary œdema. Signs of cardiac inadequacy are present at all times, and yet she shows no traces of dropsy or special venous congestion. In both of these cases hypertrophy still predominates, and is able to endure the high endocardial blood-pressure so long as this is not intensified by the strain of physical effort. The nocturnal seizures in the second lady were probably due to the augmentation of blood-pressure in the arteries occasioned by the recumbent position in sleep. This at length overpowered the left ventricle, which temporarily became weaker than the right, and acute pulmonary œdema supervened. In some of these cases of chronic myocarditis such attacks form the principal feature, and the cases are particularly grave on this account.

Cases of Secondary Valvular Insufficiency.—Lastly, one occasionally meets with cases of myocardial degeneration which masquerade in the guise of a mitral or aortic regurgitation. I do not refer to instances of atheromatous valvular disease, but to those in which the valvular incompetence is relative. Arthur R. Edwards has reported a case of relative aortic insufficiency from extensive myocardial degeneration, and I have myself observed three cases in which the necropsy revealed the same condition. In all of them the clinical history was that of aortic regurgitation.

Relative mitral insufficiency may result from fibroid degeneration of the papillary muscles, but is more often due to dilatation of the ventricle. I do not now refer to Balfour's Curable Mitral Regurgitation, which is seen in chlorosis and anæmia, or to that form seen in young athletes as an effect of acute strain. These all yield to appropriate treatment. I am now speaking of left-ventricle dilatation and secondary mitral insufficiency seen in cases of chronic myocarditis. I have under observation a man of sixty-five, a veteran of the late civil war, whose mitral valve leaks in consequence of great dilatation of the ventricle. There is no history of inflammatory rheumatism or any other disease to occasion endocarditis, but there is history of severe physical effort (climbing a mountain) ten years ago. Previous to that strain he had no cardiac symptoms, but since then his mitral murmur and dilatation of the ventricle have been present. At times the murmur wholly replaces the first sound, but as the ventricle retracts under treatment by baths and resistance exercises, the first sound becomes audible and cardiac impulse palpable. I think few would venture to assert that in this case the myocardium is healthy.

I have notes of the case of another gentleman of forty who presented the signs of a typical mitral regurgitation, and who for four years struggled to preserve his compensation. He gave a history of mild inflammatory rheumatism, of gonorrhœa, and of a thrombophlebitis of the right femoral vein, and therefore his valvular incompetence was quite naturally supposed to be of endocarditic origin, a conclusion that was strengthened by the occurrence of two attacks of subacute articular rheumatism during the time he was under observation. He at last died, after having been confined to his bed for only a week, with symptoms of cardiac exhaustion. There was no œdema, very insignificant venous stasis, and at first a profound sense of weakness rather than of shortness of breath. Towards the close of his illness, however, dyspnœa asserted itself, becoming rather spasmodic. His temperature grew subnormal, the pulse feebler and slightly more rapid, and he died apparently of simple cardiac asthenia.

The necropsy made by Dr. W. A. Evans disclosed a perfectly healthy mitral valve, and on the tricuspid, changes too insignificant to have affected their function. The myocardium was intensely fatty, particularly of the right ventricle; the cavities were all more or less dilated. The coronary arteries were healthy, but the aorta was congenitally small throughout. This man had been an athlete in college, and subsequent to his death I learned that before his symptoms of cardiac inadequacy began he had overstrained his heart by a long, hard bicycle ride. Owing to the apparent integrity of the coronary arteries in this case, I believe there can be only two explanations of his myocardial decay. It was either an expression of chronic myocarditis in the strict sense of toxic origin, or of a disproportion between the work required of it and its nutrition, this latter being restricted by reason of the congenital smallness of the aorta, which also had served to put undue strain upon the myocardium.

Finally, in concluding what I have to say upon the symptomatology of myocardial inadequacy, I desire to add a few words concerning two symptoms which are generally thought indicative of fatty degeneration of the heart. These are yawning and sighing. I have never, however, been able to satisfy myself of the import of these two symptoms. Indeed, I not only have seen many cases of myocardial inadequacy in which they were absent, but I have, on the other hand, observed them in patients who presented no suspicion of myocardial disease, as in young neurotic or anæmic women. I should certainly attach no value to yawning and sighing in the absence of other less doubtful symptoms, and in suspected cases of cardiac degeneration I should esteem them of very minor importance.

Physical Signs.—*Inspection.*—In most cases inspection is negative. If the eye detects signs of stasis, there is nothing in this fact to indicate the underlying condition. The general appearance of the individual may show to the experienced physician signs of premature or senile decay. When hypertrophy of the left ventricle is present, this may be shown by the displaced apex-beat. But in the class of cases in which it is the most difficult to arrive at a definite conclusion—that is, middle-aged and well-preserved men with capacious chests, the cardiac impulse is not visible because of the chest-capacity and lung-volume. Consequently, it may be said that the chief value of inspection lies in the fact of its negativeness, for other disorders of the heart than myocardial degeneration are very apt to furnish some visible indication of their nature.

Palpation.—This is of value in the determination of œdema and of hepatic engorgement even more than in the examination of the heart. Yet by careful palpation of the præcordium one is often able to locate an apex-beat which is too feeble to be visible. It may enable one also to perceive that the cardiac impulse has the diffused jogging character of dilatation with hypertrophy, or the feeble, slapping shock of dilatation. Palpation is of special value in disclosing the state of the arterial coats. If these feel thick and resisting, or tortuous and uneven, like a string of beads, they furnish presumptive evidence that the heart-muscle is not sound. In searching for signs of cardiac incompetence one should always endeavour to palpate the liver. If the lower border of this organ can be felt below the costal arch, and particularly if it is smooth, rounded, firm, and perhaps tender, there is hepatic congestion, probably secondary to more or less cardiac inadequacy.

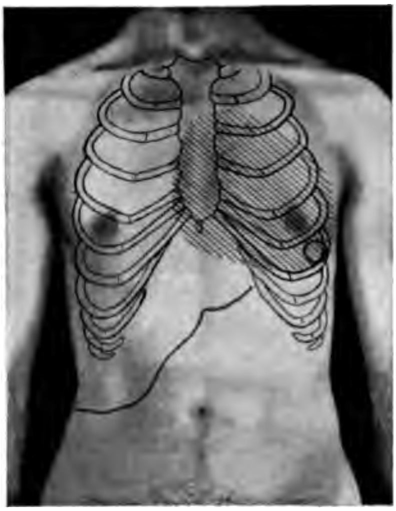


FIG. 103.—SHOWING SHAPE OF RELATIVE DULNESS IN HYPERTROPHY. Quadrilateral with rounded corners.

Percussion.—This means of investigation should never be neglected, for very much depends upon the size of the heart. Absolute dulness may or may not be increased, but as the organ is enlarged in most cases of myocardial degeneration, careful percussion usually elicits an augmentation in the area of cardiac dulness. If this is found increased to the left and upward, it indicates left-ventricle hypertrophy; if to the right and downward, enlargement of the right ventricle. In general cardiac hypertrophy the area of deep-seated dulness is of a quadrilateral outline with rounded corners (see Fig. 103).

In estimating the size of the heart it is customary to take the left verticle nipple-line as the normal boundary of deep-seated dulness at the left. But Fraentzel dwells particularly on the liability to error existing in the custom of considering the left nipple as the normal boundary of relative dulness on that side. If the dulness is not found to pass beyond this mark it is taken for granted that the size of the heart is normal. It should be remem-

	Age.	Weight.	Height.		Circumference of chest.	Distance from sternum to left nipple.
			Feet.	Inches.	Inches.	Inches.
K. A.	29	155	5	7	33	3¼
T. B.	24	135	5	7	34	3
C. B.	25	130	5	7	34	3½
E. B.	30	150	5	9	34	3¾
W. C.	28	145	5	6	34	3¼
S. D.	29	157	5	9	35½	3¼
H. De V.	42	191	6	1	40¼	3¾
R. E.	24	149	5	6	36	3½
E. E.	24	185	5	7	36	3¼
S. E.	33	170	5	8	36	3½
F. G.	23	160	5	10	34	2¾
G. G.	33	125	5	4	30	3½
C. H.	28	161	5	9	35	3¼
H. J.	23	195	5	10	40	4
P. J.	23	158	5	7½	35	3¼
G. L.	31	145	5	9	34	3¼
L.	?	155	5	9½	37	3
F. M.	23	140	5	10	33½	3
J. M.	21	130	5	7½	33	2⅞
H. M.	82	165	6	1	35	3½
R. S.	29	164½	5	10½	35½	3½
J. S.	30	135	5	4	33	3
J. S.	30	162	5	9½	38	3¼
W. V.	25	189	6	0	39	3½
J. W.	25	204	6	0	39½	4⅛
J. W.	27	150	5	7½	38	3¼
J. W.	24	130	5	6	33	3
C.	22	145	5	8½	35	3¼
C. H.	43	189	6	0	39	3½

bered, however, that the distance between the midsternal and left mamillary lines is by no means always the case. I have not infrequently found the left nipple situated 5 inches from the midsternum. Measurements of twenty-nine of my students, taken for the purpose of determining variations in this regard, gave the results shown in the table on the opposite page.

These figures indicate plainly that the only accurate means of determining the boundaries of the heart by percussion lies in measuring the distance to which the area of deep-seated dulness extends to the left of the median line. The size of the normal heart, as shown by percussion, has already been stated in the introductory chapter.

One often obtains valuable information by the sense of increased resistance on firm percussion, and hence the value of Ebstein's palpatory percussion. I am in the habit of verifying the results of percussion in the ordinary way, by recourse to auscultatory percussion, and am frequently surprised and gratified to see how closely they correspond.

Auscultation.—Here, too, much depends upon the thickness or thinness of the chest-wall. If hypertrophy exists the first sound at the apex is prolonged and of low pitch, while the second is usually clear and ringing. In some cases the systolic sound is muffled and indistinct. At the base of the heart the aortic second is sharply accentuated, ringing, or it may be so intense as to be actually banging. There is also intensification of the pulmonic second sound when the left ventricle begins to fail, and at the base of the heart one sometimes detects reduplication of the second sound. If the first in the region of the apex is short and sharp, resembling the normal second, it indicates dilatation rather than hypertrophy.

Occasionally the heart-sounds take on the canter-rhythm described at length in the introductory chapter. This characteristic rhythm is limited to one or the other ventricle, and therefore to the neighbourhood of the left nipple. It is especially likely to appear in left-ventricle dilatation consequent upon a granular kidney, but, according to Fraentzel, occurs, although rarely, in enlargement of the heart from other causes. This gallop rhythm must be kept distinct from reduplication of the second sound heard at the base, and from that apparent or simulated doubling of the second sound that is not infrequently discovered in the mitral area in cases of stenosis of that orifice.

A full, tense, not accelerated pulse, a dull first sound, and an accentuated aortic second, form a combination of signs highly suggestive of hypertrophy of the heart, even though its area of relative dulness cannot be defined with certainty.

There is no pathognomonic sign of degeneration from coronary sclerosis, and often the heart-sounds appear normal. It is highly important, however, and sometimes yields valuable information, to study the relative pitch and intensity of the several sounds. If, as there is good reason to believe, one of the elements entering into the make-up of the first sound is a muscular element, imparting to the sound its booming quality, and caused by contraction of the ventricular and papillary muscles, then impairment of their contractility through disease should theoretically diminish the intensity of the first heart-sound. Experience shows that this is precisely what takes place in some instances. Over the weakened left ventricle this sound at the apex may be weaker than that over the right ventricle, having a distant or muffled character. The pitch of the sound may be raised also and its duration somewhat shortened. The second sound at the apex is often relatively louder than the systolic, and on moving the stethoscope to the base of the heart this intensification of the second sound is found due to accentuation of the aortic second, which may even possess a ringing character from sclerosis of the aorta. In other cases the second sound at the right of the sternum is feebler than that in the pulmonary area.

As the aortic second sound should be the louder of the two in persons of the age at which myocardial degeneration usually develops, relative weakening of the second sound in the aortic notch points to diminished vigour in ventricular contractions, and hence furnishes indirect evidence in favour of degeneration.

Murmurs are accidental findings, and are due either to relative incompetence of the valves or to atheromatous roughening of the orifices. In either event they may afford valuable testimony as to the state of the heart-muscle. A murmur may, of course, in some instances be the result of a rheumatic valvular defect, as will be shown by the history.

Diagnosis.—From the foregoing, it is evident that in the diagnosis of degeneration of the myocardium, but limited information is derived from a study of the heart. There is no form of cardiac disease, therefore, in the diagnosis of which so much depends on the judgment and experience of the physician. In valvular defects there are murmurs to serve as guide-posts; in hypertrophy or dilatation there is obvious alteration of size. In the affection under consideration the volume of the organ may or may not be changed, and therefore great dependence must be placed on age, state of the vessels, history, and symptoms.

Age is so important an etiological factor that the development of cardiac insufficiency in an individual well on in years may be set down to degenerative changes with tolerable certainty. It is quite otherwise when heart-weakness, without obvious signs of disease, develops in a person about the middle period of life. In such persons careful search should be made for traces of premature decay, for indications of renal disease, or a gouty diathesis, etc. There is an old saying that a man is as old as his arteries, and therefore the radials, temporals, and other peripheral vessels should be carefully palpated for evidences of thickening or for nodular deposits of lime-salts.

It may be necessary in some cases to make an ophthalmoscopic examination of the retinal artery for the signs of sclerotic change which are said to first manifest themselves in this situation. The physician should note the appearance or not of premature whitening of the hair, and examine the texture of the skin. I have more than once observed that persons with a strong suspicion of fatty degeneration of the heart have a skin that has lost its elasticity and feels peculiarly soft, as is often the case in the aged.

The examiner should scrutinize the fingers and ears for chalky deposits, and the nails for those longitudinal ridges said to be indicative of the gouty state. In this way valuable hints may often be obtained.

The *urine* should be analyzed carefully, and repeatedly if necessary, for evidence of nephritis, since it is well known that degeneration of the myocardium is a frequent accompaniment of chronic renal disease, particularly the interstitial form.

Minute inquiry into the patient's history may elicit facts concerning family tendencies, personal habits, previous diseases, etc., that may throw light upon the nature of the present malady. It is particularly important to ascertain whether the patient has suffered from attacks of angina pectoris or cardiac asthma. The significance of the former in individuals past middle age is very different from that of anginoid seizures in adults under forty, especially women.

Even in spite of the most painstaking investigation and attention to all circumstances, however trivial, a positive diagnosis in this class of cases is not always possible without awaiting the results of therapeutic management. If decay of the heart-muscle is present, it will be ultimately shown by the gradual or more rapid development of symptoms sufficiently characteristic to settle the diagnosis.

Aneurysm of the heart is only possible of diagnosis when it is of sufficient size to affect the outline of cardiac dulness in a way to suggest localized bulging of the heart-wall. It is stated that cardiac aneurysm may be suspected when there is a striking disproportion between the force of the cardiac impulse at or near the apex and the smallness and feebleness of the pulse. Its existence can probably be determined by fluorescopic examination.

In most cases of *cardiac rupture* its occurrence can only be suspected but not determined before the death of the patient. It may be surmised in cases running the extremely acute course described in Symptoms. Physical signs pointing to fluid distention of the pericardium, with a pale, anxious countenance, a small, feeble, irregular, it may be intermittent pulse, and other symptoms of profound shock, furnish strong evidence that rupture of the heart-wall has taken place. If life is sufficiently prolonged a correct diagnosis is often possible, but when death occurs within a few minutes the physician can rarely do more than conjecture the occurrence of rupture.

The diagnosis of chronic myocarditis is largely a matter of probabilities, since there are no pathognomonic signs of the condition. Physical examination may disclose certain gross changes, as hypertrophy or dilatation, or a combination of both, and pathology teaches that such hearts are as a rule more or less degenerated, but we possess no means of determining outside the dead-house to what extent the heart-muscle is diseased or the precise nature of its degeneration. The majority of elderly individuals who consult us because of cardiac symptoms do not suffer from the consequences of rheumatic endocarditis as do the young. They present evidence of cardiac incompetence; of this we can be certain, but concerning the state of the myocardium we must take much for granted.

Prognosis.—This depends upon the cause, the degree of the hypertrophy, and the state of the heart-muscle. If the high pulse-tension is due to luxus consumption, and the individual is young and robust, correction of his habits may lessen peripheral resistance, and may retard, if not wholly prevent, development of cardiac inadequacy. In cases of advanced renal or vascular disease there are two dangers: occurrence of apoplexy and the breakdown of the heart under conditions of unwonted strain. If the cause, whatever its nature, is persistent and not amenable to treatment, the ultimate prognosis is unfavourable, because there will at length come a limit to the hypertrophy and the heart-wall will give way.

So long as the myocardium is functionally healthy—that is, receives sufficient nourishment—the hypertrophy proves a preservative measure; but when incompetence sets in, the most favourable management can do no more than defer the evil day. Palpitation, and particularly intermittence of the pulse, are unfavourable signs; they may be the first evidence that the heart is yielding to the unequal struggle, or by occasioning incomplete emptying, and hence distention of the cardiac chambers, they may hasten the coming on of dilatation.

In forming a prognosis in any given case one must take into consideration also the age and temperament of the patient, and the state of his general nutrition. The younger the patient and the greater his self-control, the better his prospects of maintaining compensatory hypertrophy and the less the likelihood of injury from excesses, emotional or otherwise. The further one gets beyond middle age the stronger the probability of the cardiac insufficiency being due to myocardial degeneration, and of the obstacle to circulation proving too much for the weakened heart-walls.

When serious symptoms at length set in there is small prospect of medical skill being able to do more than patch up the crippled heart. In a word, the prognosis depends upon the relation existing between the demands made upon the heart and its ability to respond. It is therefore almost entirely a question of cardiac nutrition. The younger the individual the less the likelihood of serious degenerative changes, but after middle age such changes are usually present and compensatory hypertrophy is rarely re-established after it has once seriously given way. One should make a careful study, therefore, of the condition of the vascular coats, as they furnish presumable indication of the state of the heart-muscle. Nevertheless, experience teaches that the latter may be extensively diseased while the arteries appear healthy. The detection of the gallop-rhythm over one or the other ventricle, most often the left, is of evil import, as it indicates a loss of muscular tone and either incipient or fully developed dilatation. In the cardiac inadequacy of chronic nephritis this symptom may be regarded as indicating a not very distant termination of the case.

In *coronary sclerosis* the prognosis is most grave. We possess no means of ascertaining the location and extent of degeneration, and hence cannot say whether life will persist a single hour. Indeed, a person with fatty degeneration of the heart-muscle can never be sure of his life from one moment to another. He may live for years, and he may die suddenly when apparently in the best of health.

The occurrence of *angina pectoris* makes prognosis doubly bad. In acute and subacute cases death is not likely to be long deferred, and except in the most acute forms, which are usually rapidly fatal, no one can venture to predict the length of life. Chronic forms of coronary sclerosis may persist for many years with ever-recurring attacks of angina. As a rule it may be stated that the more easily and frequently pain is induced the graver is the prognosis.

The *arrhythmic form* of chronic myocarditis is apt to run a very chronic course, whereas those showing attacks of cardiac asthma or of acute pulmonary œdema are in danger of terminating abruptly in such an attack. The development of *Cheyne-Stokes* respiration is in most instances an indication that the end is not far off (see article on this type of breathing). *Syncopal attacks* are likewise of evil portent, owing to the danger of sudden death from asystolism at such times.

The cardiac insufficiency of Bright's disease and diabetes is of particularly great gravity, since the abnormally high pulse-tension, which is the cause of the cardiac embarrassment, cannot be removed, and prevents the left ventricle from regaining its lost power. A serious breakdown in this class of cases, therefore, may be said to be irreparable.

Finally, the prognosis is also determined by the presence or absence of sclerotic changes in the kidneys, lungs, and liver, since the healthier these organs the less the strain upon the diseased heart. *Chronic gastritis*, with its flatulent distention of the hollow viscera, influences prognosis both through mechanical pressure and the generation of injurious toxines.

Acute bronchitis or other illnesses, in particular pneumonia and influenza, must always fill the medical attendant with alarm, since it requires but little to throw the balance one way or the other in these cases, and acute infections are very liable to prove the immediate cause of death in cases of chronic myocarditis, which, without such an intercurrent affection, might have persisted for years longer. Conditions of environment also affect prognosis, an individual who is able to spend his winters in a mild climate and avail himself of all other means of warding off injurious influences being, *ceteris paribus*, likely to live longer than he who is compelled to toil on for his daily bread.

In conclusion may be quoted Huchard's emphatic statement concerning cases of myocardial disease: "Their evolution is latent, their beginnings insidious, their course paroxysmal, their progress interrupted, their visceral complications various, and their explosions of cardiac insufficiency are sudden."

Treatment.—This must be considered first with regard to preservation of cardiac competence, and second with reference to the stage in which heart-power is either showing signs of failure or has actually been lost—pronounced cardiac insufficiency. Medical aid is not sought so long as the myocardium is adequate, and if the discovery of hypertrophy is made, it is only by accident. When, however, such discovery is made, it should be the physician's duty to call the patient's attention to the dangers threatening him in the future, and to show him how his habits of life are likely to affect his heart.

The management is now along the line of prevention; patients who habitually eat or drink too much must have the evils of gluttony explained to them, and be put upon a diet that will not overtax kidneys, vessels, and heart. The man who takes little or no exercise, and is too rapidly gaining weight, must be sent to 'the gymnasium to be put in training, or must be made to walk more and ride less.

If vessels are sound and heart still competent there is nothing better for such patients than moderate bicycling, tennis, ball-playing, etc. If such sports are thought too vigorous, there is golf, which is an ideal form of exercise, since it trains the eyes and muscles without subjecting weak organs to undue strain. Those with corpulent, flabby abdomen are much benefited by a course of massage and Swedish movements. The processes of digestion and assimilation are improved, and constipation, if present, is generally corrected.

Gouty individuals or persons suffering from defective excretion usually derive benefit from a semi-weekly or a weekly Turkish bath. This not only increases elimination, but lessens blood-pressure. This seems especially beneficial to persons addicted to the abuse of alcohol and tobacco. If the cause of the hypertrophy is not preventable, or if vascular and renal changes are pronounced, then patients should be frankly informed of their condition, and warned against undue muscular effort, or whatever may serve as an additional and unnecessary strain to the heart-muscle. Arterial and kidney disease call for still greater strictness in the matter of diet.

A highly nitrogenous dietary often serves to intensify the already existing high arterial tension, while a vegetarian diet, or one bordering thereon, lowers blood-pressure.

Digestive disturbance and constipation must, if possible, be corrected, since they not only increase arterial tension, but may produce palpitation and intermittence, which, if allowed to go on, may ultimately impair the integrity of the heart-muscle, which in this stage it is our aim to preserve.

Cardiac tonics, especially digitalis, are not needed at this time, and if administered are likely to do harm. Our attention is to be addressed not to the heart itself, but to its protection from all injurious influences.

Unless induced acutely by severe heart-strain, signs of inadequacy begin to declare themselves slowly and at first very insidiously, so that the management may be said to pass almost imperceptibly into the treatment of symptoms directly due to:

Commencing Loss of Heart-power.—Among the earliest signs of this second stage are apt to be tachycardia and palpitation. These are not to be looked upon as indications of excessive hypertrophy, for such a thing does not exist. They are the earliest token that the organ is beginning to find its work too heavy. Therefore, nothing is more pernicious than to attempt to control these symptoms by aconite or veratrum viride, which are powerful cardiac depressors.

Digitalis and strophanthus are likewise not to be always prescribed for attacks of palpitation or for tachycardia until after an attempt has been made to discover and remove possible sources of irritation and increased peripheral resistance.

In my experience a too rapid or violent action of the heart in this stage may be due to digestive disorders, constipation, or faulty excretion, which augment arterial tension, and hence often subside with the removal of the cause. To this end I find very satisfactory a periodic dose of calomel or a blue pill, followed next morning by an aperient water.

It is also necessary in these cases to restrict the diet by cutting out red meats and limiting the intake of water or other fluids at meal-time. The former raise pulse-tension by means of their extractives, while the latter distend the stomach and abdominal vessels.

Should blood-pressure still be too high, it may be reduced by one of the nitrites or a potash salt. Three grains of potassium iodide may be given after meals in essence of pepsin without disturbing the stomach, or nitroglycerin, $\frac{1}{100}$ of a grain, may be given every three hours. Erythrol is said to be more lasting in its effects on the arterioles, but this advantage has not seemed to me sufficient to compensate for its greatly increased cost.

Should such treatment fail to control cardiac action, then it is well to resort to digitalis or allied remedies. They may be given in conjunction with iron, arsenious acid, or strychnine.

Gentle exercise is now very beneficial by its action on the heart and vascular system.

It causes dilatation of the intermuscular arterioles, promotes venous flow, and thus tends to restore circulatory equilibrium, removes waste products from the tissues, and flushes the heart-muscle with freshly oxygenated blood. This explains why patients who feel præcordial oppression upon starting out for a walk often experience a sense of relief and well-being after their exercise has "warmed them up," as they say.

Gentle pedestrian exercise is to be recommended, therefore, in this stage of commencing cardiac incompetence, but under certain restrictions. Patients must be cautioned to begin their walk at a slow pace, and to increase their speed only as they find exercise and breathing grow easier. Walking against a cold or strong wind is very trying, and on such days they should walk with and not in the face of such wind. The carrying of heavy parcels is to be forbidden, and the restraint of trunk or limbs by tight clothing is inadmissible.

The ascent of stairs and hills is fraught with danger to the failing heart, and should be avoided. Oertel's plan of hill-climbing is to be advised only for patients whose hearts still retain a fair measure of their integrity and whose judgment can be relied upon. The principle underlying this mode of treatment consists in the ascent of gentle inclines at a rate of speed that does not cause dyspnœa or palpitation. Only when such acclivity can be surmounted with ease is a steeper grade to be allowed. If hill-climbing is done so as not to occasion respiratory or circulatory embarrassment, the heart-walls are gradually strengthened and a tendency to dilatation is overcome. This form of exercise requires excellent judgment on the part of the patient lest he overdo, and on the part of the physician in the selection of suitable cases.

Another kind of cardiac exercise not open to the same objection, and suited to a larger number of cases becauses its effects can be more accurately gauged, are the "resistance exercises," which were described in detail in the chapter devoted to Treatment of Valvular Disease. If golf is permitted to patients in this stage of deficient cardiac power it should be restricted to putting, or at most to the playing of a limited number of holes. Whatever the form of outdoor exercise allowed, the following restrictions should be imposed: (1) Patients must not exercise immediately after eating, the length of time devoted to rest being determined by the degree of cardiac weakness. In most cases patients should remain quiet for at least an hour, and when the heart is feeble Fraentzel does not allow exercise before three or four hours after a meal. (2) Walking or other exercise should not be indulged in to the point of fatigue. In some cases indeed it should be for only a short period, several times repeated during the day. (3) In cases showing decided indications of a threatened loss of adequacy, rest in a recumbent posture must be insisted on at the close of exercise.

As our aim at this time is to prevent the heart from becoming still more taxed in its labours, and blood-pressure is increased by hearty feeding, it is necessary to restrict the diet. It is quantity even more than quality that is harmful, and hence patients should be told to eat lightly. Too much liquid raises blood-pressure in the abdominal vessels, and therefore it is well to restrict it to 8 or at most 10 ounces with each meal. Alcoholic stimulants, if permitted at all, must be in the form of a light, dry wine, or still better of a modicum of whisky, largely diluted with water. Tobacco is to be allowed in great moderation, a small light cigar or a single pipeful of mild tobacco after meals. Huchard, Fraentzel, Krehl, and others are very strenuous in their opposition to strong Havana cigars on the ground that they augment arterial tension, and state that many middle-aged men with weak hearts find out for themselves that they are obliged to substitute mild domestic cigars for the heavy Havana ones to which they have been accustomed.

Excesses of all kinds are injurious, and these patients are to be warned against the harmful effect of frequent sexual indulgence. Indeed, the principle that must govern the daily life of these individuals is moderation in all things. If patients give due heed to the doctor's admonitions they may succeed in holding their hearts in *statu quo* for a considerable time. Unfortunately, however, the tendency of myocardial decay is downward, and hence we are called on, soon or late, to institute active treatment for the relief of symptoms which mark the arrival of the third stage.

Cardiac Incompetence Pronounced.—Venous and visceral congestion now begins to manifest itself, and calls for the more vigorous and frequent use of cathartic remedies. It is also generally necessary to resort to cardiac tonics, and of these digitalis heads the list, although strophanthus, spartein, convallaria, adonis vernalis, and caffeine are all useful. Whenever digitalis is administered to a patient who exhibits high pulse-tension, particularly if this depends on arterial thickening, it should always be given in conjunction with an iodide salt or nitroglycerin to counteract its effect on the arterioles. So long as cardiac weakness is not extreme the dose of digitalis may be small, 10 drops of a fat-free tincture thrice daily, or 15 drops every twelve hours. Given in this way it may be continued for weeks or even months without losing its effect or exhibiting its cumulative action. In more than one instance of myocardial inadequacy with stiff arteries I have seen striking results follow the prolonged use of strophanthus. It is sometimes well to combine these two remedies, a few drops of each being taken at a dose. Spartein sulphate is highly recommended by the French when the pulse is irregular, but although I have tried it repeatedly I have never been able to satisfy myself of its beneficial effect or advantage over digitalis.

Strychnine is so indispensable a heart-tonic that I believe it should be taken by this class of cardiopaths as regularly as is their food. A fortieth or even a thirtieth of a grain three times a day is not at all too much for the average patient.

The one form of treatment from which I have seen patients with myocardial insufficiency derive most benefit are the natural or artificial Nauheim baths (see page 466). They should be combined with resistance exercises. In my opinion this form of treatment is particularly adapted to this class of cardiopaths, and I have rarely seen a case of dilated hypertrophy which has not been improved by its judicious employment. I recall a typical example of this form of heart-disease in a medical man of forty-four, who began to manifest symptoms of threatening dilatation. His area of absolute cardiac dulness was greatly increased, particularly to the left, and anything more than moderate exercise occasioned a very considerable degree of discomfort. Six weeks of baths completely restored the heart's power, and although five years have now elapsed, the doctor is still able to attend to the duties of a large and exacting practice. His professional calls have required him to daily climb many flights of stairs, and although unwonted exertion still calls forth some degree of breathlessness, he has, by keeping down his pulse-tension through a somewhat restricted diet and an occasional purgative, never again displayed the same threatening symptoms. The last time I heard from him he had taken to a bicycle, and by careful riding succeeded in still further strengthening his heart. At the close of his course of baths absolute dulness had returned to normal, and the relative become manifestly reduced. He weighed over 200 pounds, and of course still has a considerable degree of cardiac hypertrophy. In the summer of 1899 I treated by means of baths and resistance exercises two middle-aged gentlemen, each with enormous hypertrophy. The symptom chiefly complained of by one was great præcordial oppression, amounting almost to what Gairdner would call "*angina sine dolore*," whenever walking was attempted about an hour after meals. His pulse was slow and tense, and his heart enormously enlarged. At the end of treatment the heart was not much reduced in size, but the sounds were manifestly stronger, and the pulse had become fuller, stronger, and slightly more rapid. He then passed a month at his summer cottage, where he daily indulged in light carpentering, and was able to ascend the sandy hill on which his home stood without experiencing the former discomfort. A very restricted diet and the daily use of small doses of the tincture of strophanthus and iodide of sodium have been rewarded by continued improvement. The second patient also derived much benefit from the baths and exercises, although, as in the preceding case, the area of deep-seated cardiac dulness did not become permanently diminished. I was frequently able to determine a reduction in the size of the heart following a bath, and he always experienced a sense of well-being and lightness in the chest. In this case there was a very obstinate indigestion, and the urine always contained an excess of solids, although it never showed albumin or casts. His pulse was for the most part irregular and intermittent, seeming to be governed in this respect by the intestinal indigestion, for every time his digestive disturbance became aggravated his pulse grew more irregular. He was subsequently induced to take a course of massage and Swedish gymnastics, with the result that not only did his corpulent abdomen become greatly reduced in size, but his digestion improved, his pulse became regular for days together, and he said he felt as well as he ever did in his life. The heart, however, still showed great enlargement.

In both these cases a change of habit as to food and exercise lowered blood-pressure, treatment of the heart restored its competence, and threatening dilatation was averted. I should add that all these three patients continued their professional and business duties while undergoing treatment.

In most cases that have reached this stage restoration of heart-power is out of the question. The problem confronting the physician is how to relieve symptoms and postpone the final catastrophe. In such, exercise is likely to do harm instead of good, and yet my experience has convinced me that harm is also likely to result from a too rigid enforcement of rest. If the breakdown is complete, the sufferer may be forced to remain in bed or his easy chair. If things have not reached this pass, I believe it is better to allow the invalid to move quietly about his room that venous circulation may be aided by muscular contraction and the deepened respiration consequent upon this exercise. In some instances venous circulation may be assisted by gentle massage, and carefully conducted resistance exercises may, by dilating the arterioles, and thus flushing the muscles, help to unload the overdistended heart.

Romberg and Fraentzel are both emphatic concerning the injury of too strictly enforced rest in chronic myocarditis, and experience has convinced me of the soundness of their advice. In the earlier years of my practice I used to consider prolonged rest indicated in all cases of cardiac incompetence from whatever cause, but I ultimately found that elderly individuals, who were not suffering from valvular disease, showed an acceleration of their downward course when they were denied all exercise and kept rigorously in bed.

The aggravation of symptoms thus resulting is attributed by Romberg to the enervating effects of inaction, the same as is observed in the case of the voluntary muscles from disuse. The same thing is observed in previously healthy persons who are obliged to remain in bed a long time, from one cause or another; when again permitted to get up they not only find their legs weak, but the first attempt at walking produces slight shortness of breath and acceleration of the pulse. This explanation is in accordance with that given by Fraentzel, and is doubtless correct so far as it goes. In the case of a degenerative heart, there is another reason which I think holds good. When a patient remains quiet in a recumbent position the venous circulation is deprived of two factors of great importance in its maintenance. These are muscular effort and deepened respiration. Muscular contraction aided by the venous valves exerts a pumping action on the venous current, and also the flow within the absorbents. Abolish the use of the muscles and you remove one of the well-known causes of venous circulation. Furthermore, with rest in bed the patient breathes more slowly and superficially, and hence blood is less rapidly aspirated out of the great veins into the right heart, and a second important factor in maintaining venous flow is diminished.

The work of maintaining the circulation now devolves upon the heart even more than under normal conditions. It must contract more powerfully that its driving force may be felt throughout the entire circle propelling the blood onward in the veins. In those cases in which breathlessness on effort is a pronounced feature absolute rest for a time may bė beneficial, *but it will not do to let these patients remain quiet for too long a period.*

The heart-muscle is weak, and cannot be left for too long a time to cope unaided with the labour of maintaining adequate blood-flow. Instead, therefore, of complete rest, it is better that the physical repose be interrupted by short periods of gentle exercise. This last, however, is to be *strictly controlled.* The patient must only be allowed to walk about his room, or at the most into the adjoining room. Under no circumstances is he to be allowed to climb stairs nor to walk about soon after a meal. He must be impressed with the danger of jumping up suddenly and of hurrying across the room. If very weak, and dyspnœa is considerable, he had better lean upon the arm of an attendant while taking his exercises.

The patient should also not be allowed to dress himself unaided, and he must be instructed not to strain at micturition or defecation, since, according to Sommerbrodt, straining during such acts raises blood-pressure reflexly, and has more than once caused sudden diastolic arrest of the left ventricle.

If massage is employed it must not be applied to the abdomen, unless very cautiously, since it raises blood-pressure. Also, if resistance exercises form a part of the management at this time, those are not permissible which constrict the abdomen or necessitate the elevation of the arms to a level above the patient's head, as they are likely to occasion dyspnœa and do harm.

In the matter of food, it is well to remember that these patients require a relatively small amount of nourishment on account of the enforced inactivity of their lives. They should consume a limited quantity of fluids, since it is an easy matter to ingest more than can be excreted by the kidneys because of congestion, and only such an amount is to be allowed as is found by actual trial to promote the renal function. Special care is to be had in ordering such foods as do not induce flatulent distention of the stomach and bowels, and when this occurs it must be relieved by carminatives and medicines that assist feeble digestion.

Of equal importance with the prevention of fermentative indigestion is the correction of constipation. This is injurious not only because it necessitates straining at stool, but also on account of its raising arterial blood-pressure, and thereby increasing dyspnœa, and the liability to attacks of angina pectoris and cardiac asthma. The patient should therefore take a laxative pill at bedtime, containing some of the well-known combinations of aloes, cascara, podophyllin, rhubarb, and colocynth, or a morning draught of some aperient water. It will not do to purge these patients repeatedly and violently, since there is danger of augmenting the already existing debility, and yet a considerable degree of hepatic engorgement may render necessary an occasional sharp purge.

Having in this way endeavoured to remove or lessen the various conditions which may embarrass heart-action, the medical adviser should next turn his attention to those therapeutic measures which usually afford a prospect of strengthening the heart-muscle. Digitalis is the agent usually employed in this as well as other forms of cardiac debility. It should be prescribed, however, with care and judgment. If the heart-muscle is greatly damaged, it is not likely to respond to the remedy, which will then exert itself chiefly on the arterioles. Through contraction of the latter blood-pressure is raised, so that instead of strengthening the heart digitalis may actually increase its labour. If given in such a case, the remedy should be prescribed in moderate doses, 5 or 10 drops of the tincture, twice or thrice daily, and its constricting effects on the vessels should be counteracted by nitroglycerin.

In coronary sclerosis the nature of the degenerative changes that take place in the heart precludes the possibility of doing anything more than to relieve symptoms. In the most acute manifestations of the disease, the physician, who has been hastily summoned, is usually able to do no more than attest the fact of death. In the somewhat less acutely fatal cases with manifestations of profound shock stimulation is urgently indicated. Time should not be lost by sending for some favourite stimulant, but use should be made of whatever is at hand. A tablet of nitroglycerin, with which every physician is usually provided, may be placed upon the tongue; and while an attendant follows this with $\frac{1}{2}$ an ounce of whisky or brandy, the physician should inject under the skin $\frac{1}{8}$ of a grain of morphine. Twenty drops of spirits of camphor, or $\frac{1}{2}$ a drachm of aromatic spirits of ammonia, properly diluted, is also an efficient stimulant, and may be repeated at intervals of twenty minutes. Meanwhile, members of the family should fill bottles with hot water and place them about the body and limbs of the patient, who is then to be wrapped in blankets. A hot bag or bottle should also be placed at the præcordium.

By these and other means every attempt is to be made to restore the failing circulation. In some cases, unfortunately, all efforts are unavailing, but should the patient rally somewhat, stimulation is to be continued in such doses and at such intervals as will maintain the heart's action. If after the lapse of a few hours it be thought best to administer food to the patient, this should be liquid and hot, as a cupful of soup or hot milk, to which the ammonia, whisky, or brandy may be added. It may now be well to order strychnine hypodermically, in doses of $\frac{1}{40}$ or $\frac{1}{50}$ of a grain every two or three hours; but digitalis, strophanthus, and the like are contra-indicated or are to be given cautiously.

If the initial symptom is an attack of angina pectoris, nitroglycerin, 1 minim, and $\frac{1}{4}$ of a grain of morphine under the skin, will probably afford relief, and may be followed by hot whisky, and hot applications to the extremities and præcordium, the subsequent use of stimulants being left to the judgment of the attending physician.

In *subacute cases*, which run their course in a few weeks, or at the most in a few months, the serious changes which the heart-muscle has undergone place the restoration of compensation out of the question. Both the physician and patient must concern themselves with such measures as tend to make the downward career as slow as possible. What strength the heart still retains must be carefully preserved, and all unnecessary demands upon it studiously avoided.

The management of chronic cases of coronary sclerosis is also largely preventive and symptomatic, but there is often enough reserve power left in the organ for considerable improvement following measures calculated to reinstate the heart-muscle to a limited degree. Exercise and diet must be governed by the same rules that apply to the more severe cases, although as time progresses and the heart appears to gain strength these restrictions do not need to be so rigorously enforced. The patient should be made to understand, however, that upon his obedience to the physician's instructions, and his care in avoiding unwise effort as well as excesses in eating or any other kind, depends his hope of prolonging life. In such cases more depends upon habit and daily routine than upon remedies. It is often interesting to observe how true it is that these patients can only learn by personal experience the wisdom which their physician has vainly tried to teach them. They may be repeatedly and emphatically warned against infraction of rules of diet and exercise, lest they thereby bring on a paroxysm of angina pectoris, or aggravate the already existing dyspnœa, and yet so soon as symptoms that serve as a monitor have become lessened, they think they can allow themselves more latitude and commit some indiscretion.

A speedy return of angina or cardiac asthma brings them to their senses, and they are again ready to submit to any restriction. The going and coming of these patients must be ordered by their medical adviser, who therefore should keep them under surveillance that he may discover early signs of impending trouble, and take prompt action accordingly.

Attacks of cardiac asthma are most surely and quickly alleviated by hypodermic injections of $\frac{1}{8}$ of a grain of morphine combined with $\frac{1}{200}$ of atropine. A prompt effect is more surely obtained by throwing the medicine into the arm instead of the leg. The relief thus afforded is sometimes almost miraculous within a few minutes, the patient being able to lie down and fall into a refreshing slumber. In the less severe forms of cardiac asthma the administration of the morphine at 10 or 11 P. M. will often carry the sufferer through the night without one of his dreaded attacks. The remedy is never so efficiently administered in any other way as under the skin, and the dose should be as small as will produce the desired effect. This will rarely be less than $\frac{1}{8}$ of a grain, which dose should be administered nightly without increase. Should it be thought best for any reason to withhold this remedy, then insomnia may be overcome by paraldehyde, chloralamide, and bromide together, or by sulphonal.

The treatment of cardiac asthma is of a necessity that of the paroxysm and the protection of the patient against influences which may precipitate an attack. If Cohnheim's explanation of its mode of production is correct—namely, that it is the result of temporary increase of left-ventricle weakness, in consequence of which its systoles are relatively feebler than those of the right—then efforts must be directed to the protection of the degenerated left heart against conditions which by raising arterial tension tend to overpower the left ventricle. Blood-pressure may be injuriously raised by the horizontal position of sleep without other factors, and the augmentation of peripheral resistance thus induced serves to overstrain this relatively too weak portion of the heart. We cannot abolish the need for sleep, nor can the patient be required to pass his nights in an easy chair, but we can guard him against other harmful influences, as constipation and flatulent distention of the bowels. The former raises blood-pressure in the aortic system; and the latter exerts injurious pressure upon the weakened heart.

Dropsy is to be combated in the usual way, by an infusion of digitalis or diuretin-Knoll, as laid down in the treatment of œdema from valvular disease.

The physician is not infrequently called to see a patient suffering from excessive distention of the right heart, consequent it may be upon the rapid giving way of hypertrophy. Two lines of treatment are open to him: a resort of free catharsis by some one of the drastic purgatives, as elaterium, or to venesection. There is no doubt of the speedy relief often following the abstraction of 16 to 20 ounces of blood from the arm, and if the urgency of the symptoms or the patient's exhaustion make the medical man hesitate to administer a drastic cathartic, no objection can be urged against the opening of a vein and the letting of blood. The relief thus afforded is justification enough for the procedure.

Moreover, this operation can then be followed by the administration of elaterium, jalap, or any other hydragogue cathartic.

In a case of extreme dilatation of the heart, seen for the first time it may be, in this dire condition, the administration of large doses of digitalis, before having depleted the heart and venous system by venesection or hydragogue catharsis, is *bad practice.* Thus overdistended, the heart cannot respond to the drug and only struggles vainly to perform its work, like the poor horse that in response to blows strives in vain to draw the too heavy load up hill. So it is with the overburdened heart. It may be better in some cases to administer diffusible stimulants, as ammonia, camphor, ether, and the like, before prescribing digitalis, and only order the latter after the pulse has been improved in strength and volume by ammonia, etc.

Dropsical accumulation in the serous cavities may be withdrawn by aspiration, often to the relief of the sufferer. Such a procedure is of course not calculated to help permanently; it may, however, by lessening pressure for a short time, enable the heart to respond to stimulation. When at length all measures have been tried and found of no permanent benefit, the medical attendant may then resort to opium in some one of its many forms to lessen the patient's sufferings. If we cannot promote restoration to health, we are justified in producing euthanasia. It is a physician's duty to prolong life, I presume; but I have seen patients kept alive for days by drugs when it seemed to me it would have been far kinder not to prolong the struggle after it became manifest that the end was not far off.

CHAPTER XXI

HYPERTROPHY OF THE HEART

Morbid Anatomy.—In hypertrophy the heart-muscle is increased in thickness and weight. Hypertrophy of the organ as a whole is judged by its weight, while that of a single chamber is better estimated by a measurement of the thickness of its walls. This increase in size seems, according to the latest investigations, to be dependent on an increase in the size of the individual muscle-fibres. According to Gutch, the increase in breadth of the fibres is insufficient to account for the total increase in weight of the organ. He thinks, therefore, that the discrepancy can be explained by taking into consideration the increase in interstitial fibrous tissue that is almost always present in hypertrophied hearts, and also by the supposition that there is, with the increase in width of the fibre, a corresponding increase in length. These two factors he considers sufficient to account for the increase without supposing any numerical increase in the fibres, and indeed evidence of the latter is wanting. The question can, however, hardly be considered settled as yet.

The hypertrophied muscle is firm, cuts with increased resistance, and is usually of a deep-red colour. Increase of muscular tissue without any corresponding increase in the blood-supply causes retrograde changes to be common in hypertrophied hearts, and in consequence yellowish streaks of fatty degeneration, or gray or whitish areas of local fibrosis, are not uncommon. This is seen especially in the hypertrophy accompanying arteriosclerosis and renal disease, in which affections the blood-supply to the myocardium may be reduced by reason of narrowing of the coronary vessels.

The normal heart weighs about 300 grammes (10 ounces) in the male and 250 grammes (8.5 ounces) in the female. These

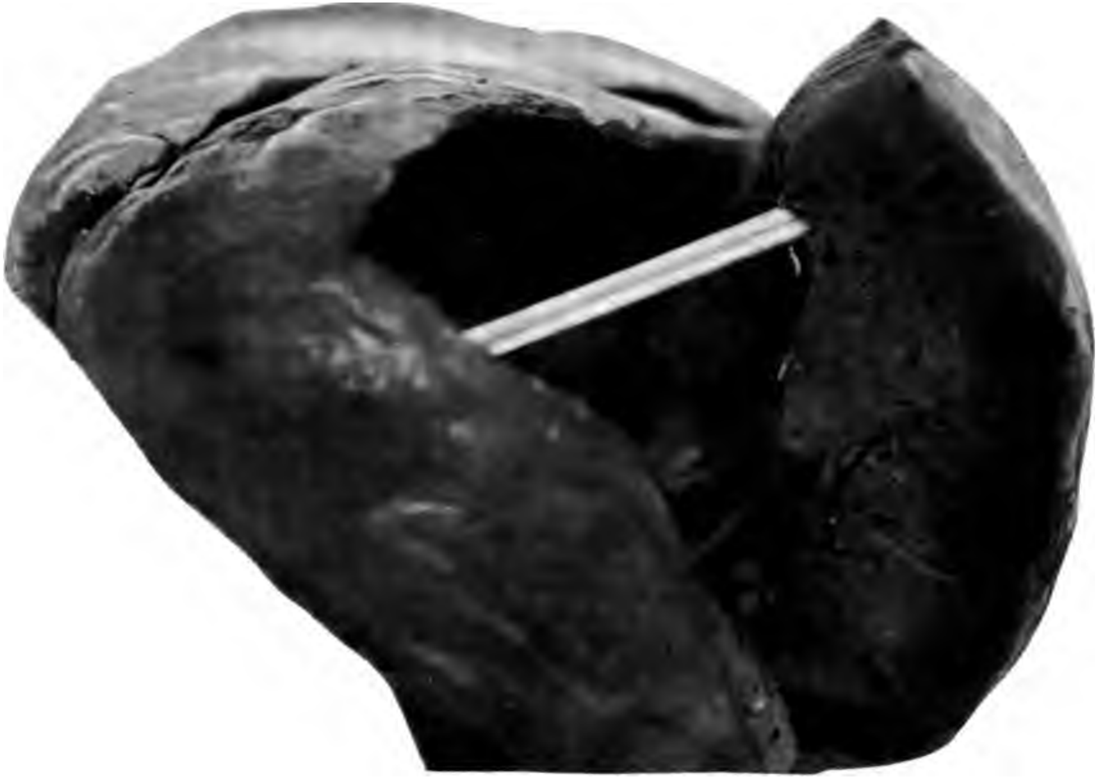


FIG. 104.—HEART SHOWING LEFT VENTRICULAR HYPERTROPHY.

figures are for individuals of the average size, but of course the heart weight varies with that of the whole body. In hypertrophy the weight may be doubled or even tripled. Stokes is said to have reported a heart weighing 66 ounces, but one weighing more than 600 grammes (20 ounces) is a very large organ. According to Eichhorst, a generally enlarged heart may attain such dimensions as to extend from the right mamillary to the left midaxillary line. When the left ventricle is chiefly involved the organ is conical and its apex blunt and broad (Fig. 104). When the right chamber is also enlarged it assumes a more quadrangular form, and the apex is formed wholly by the right ventricle (Plate III).

The papillary muscles and columnæ carneæ share in the general hypertrophy, the latter especially in the right chamber (Osler). Hypertrophy may be circumscribed, however, and then the trabeculæ, papillary muscles, either conus, or one of the auricular appendices, may be the seat of the change. Such local hypertrophy is not common and probably is due to trophic changes rather than to any circulatory disturbances.

Post-mortem rigidity of the heart-muscle may simulate hypertrophy by causing the heart-wall to be thicker than normal, and hence increase in weight is the only trustworthy sign.

Concentric hypertrophy, or thickening of the wall of a chamber without increase in its capacity, is but rarely met with (Fig. 10). It is usually the result of stenosis. Hypertrophy combined with more or less dilatation of the chamber—i. e., eccentric hypertrophy—is by far the more usual condition. When such a heart comes to autopsy, the dilatation has, as a rule, broken down the hypertrophy and is the predominating feature.

For the purposes of comparison, I give the following figures, quoted by Eichhorst, from Thoma's tables:

Weight of Heart

Until the end of the first year	37	grammes.
Second to fifth year	50 to 70	"
Sixth to tenth year	70 to 115	"
Eleventh to fifteenth year	130 to 205	"
Sixteenth to twentieth year	218 to 254	"
Twenty-first to thirtieth year	260 to 294	"
Thirty-first to fiftieth year	297 to 308	"
Fiftieth to sixty-fifth year	308 to 332	"
Sixty-fifth to eighty-fifth year	332 to 303	"

Eichhorst also furnishes the following table from Bizot:

	In males.	In females.
	Millimetres.	Millimetres.
Length of the heart	85 to 90	80 to 85
Width of the heart	92 to 105	85 to 92
Thickness of the heart	30 to 35	30 to 35
Thickness of the left ventricle at the base	10.1	9.8
Thickness of the left ventricle at the middle	11.6	10.8
Thickness of the right ventricle at the base	4.5	3.7
Thickness of the right ventricle at the middle	3.1	2.8
Thickness of the right ventricle at the apex	2.5	2.1
Thickness of the septum ventriculorum at the middle	11.0	9.0

Etiology.—Hypertrophy of the heart is rarely met with clinically except as secondary to some other condition. Thus we have seen that it is a part of the process styled chronic myocarditis, and is present also in valvular disease and adherent pericardium. It is often, though not necessarily, associated with arteriosclerosis. These affections may all be ranked among its etiological factors, but, as it is not of hypertrophy thus occasioned that I intend now to speak, they may be dismissed with this bare mention.

Hypertrophy of the whole heart, but chiefly of the left ventricle, is an almost invariable sequel to chronic disease of the kidneys, especially of interstitial nephritis, although also of the chronic parenchymatous variety and, according to Fraentzel, of long-standing pyelonephritis. There is persistently high pulse-tension in these cases, but there is probably some additional influence at work in the production of the hypertrophy—toxæmia, it may be, or atheroma. Cardiac hypertrophy of this origin becomes a clinical entity only as it is incidentally discovered in connection with the nephritis or after indications of myocardial inadequacy have declared themselves.

A much less frequent but by no means unimportant cause of hypertrophy of the left ventricle is congenital smallness of the aorta or of the entire arterial system. There is usually some other abnormality, as persistence of Botalli's duct, whenever the narrowing is limited to the isthmus of the aorta or is extreme; but in minor degrees of narrowing an increase in the thickness of the heart-wall is the only result. This condition is not unimportant, for, according to German authors, it leads to cardiac incompetence and dilatation in young soldiers who are subjected to the strain of long, toilsome marches.

In chronic Bright's disease and congenital smallness of the aorta, hypertrophy develops in consequence of abnormal peripheral resistance, which forces the heart-muscle to perform extra work. It therefore becomes increased in size (see Morbid Anatomy), the same as does a skeletal muscle under like conditions. Yet the muscular fibres could not grow in thickness and length if they did not receive sufficient nourishment, and hence augmented nutritive supply is indispensable. It is this consideration which leads German writers to regard the consumption of inordinate quantities of beer as an undoubted cause of the enormous hearts seen among excessive beer-drinkers of Bavaria. The intake of large amounts of fluid alone would not be capable of producing cardiac hypertrophy, no matter how greatly they increase peripheral resistance, but containing no inconsiderable proportion of nutritive elements, as it does, the excessive consumption of beer furnishes all the requisites for the causation of hypertrophy.

It is believed that contestants for athletic honours, particularly oarsmen and professional bicyclists, develop this form of heart-disease, and doubtless some of them do. The hypertrophy is thought to result from the heart having to overcome abnormal peripheral resistance created by severe muscular effort. This is not the correct explanation, since the initial rise of blood-pressure occasioned by muscular contraction is later on followed by a fall as the intermuscular arterioles become dilated. Therefore some other factor is responsible for the hypertrophy. This may lie in some unrecognised abnormality of the vascular system, but in the case of professional wheelmen and rowers is probably due to the constrained position they take during their exertions. As seen in the next chapter, the heart-strain of athletic contests is much more likely to result in dilatation.

In the case of soldiers, mountaineers, peddlers, labourers, or others who carry heavy packs strapped on their shoulders, the injurious effect on the heart is to be attributed to the combined influence of respiratory embarrassment and arduous physical exertion, even granting that there are no such influences at work as abuse of alcohol, atheroma, etc.

Rapid and violent action of the heart of a psychical origin is also thought to produce hypertrophy, but it is likely that tachycardia and palpitation alone are incapable of such a result. The hypertrophy and dilatation of the heart observed in exophthalmic goître is to be attributed not to tachycardia but to the underlying condition, whatever that may be.

Hypertrophy of the right ventricle is a sequel of various thoracic disorders—i. e., pulmonary emphysema, cirrhosis of the lungs, pleuropericardial adhesions and chest deformity, as kyphoscoliosis. In these conditions there is excessive peripheral resistance in the lesser circulation from compression or even obliteration of pulmonary capillaries. In time, as the nutrition of the heart suffers, its undue strain leads to dilatation and even to degeneration.

A so-called physiological hypertrophy of the left ventricle is said, especially by the French, to take place during pregnancy. There probably does develop an increased weight of the heart, a true hypertrophy of the muscle-fibres, but it is never so considerable as to become of clinical importance.

To sum up: As remarked by Krehl, the development of hypertrophy has to do essentially with the propulsion of an increased volume of blood, with the overcoming of abnormal resistance, or with a union of both these factors, and each one of them may depend upon a variety of causes.

Symptoms.—Hypertrophy of the heart is to be regarded as a wise provision on the part of Nature by which the organ is enabled not alone to perform increased work but to accommodate itself to those conditions which render increased work necessary. The normal heart can perform increased labour by putting forth extra exertion, but its ability in this direction is limited. If, therefore, the heart did not respond to demands for extra effort by the development of hypertrophy, its accommodative power to diseased conditions would soon reach its limit. Consequently cardiac hypertrophy may be regarded as a conservative process.

These considerations make it apparent that there are no symptoms directly referable to hypertrophy of the heart as such. If tachycardia, attacks of palpitation, and irregular or intermittent action of the organ disturb the patient, they are not due to the increase in its size but to disturbing conditions without, or are to be regarded as an indication of beginning inadequacy. In other words, the extra labour required of the heart is beginning to tell on it, and if the undue strain is continued, its reserve strength will become exhausted. The hypertrophy is discovered either accidentally or because a supervening dilatation occasions subjective sensations which bring the individual to the physician.

Physical Signs.—*Inspection.*—The amount of information derived from inspection of the patient depends upon the degree of hypertrophy and conditions residing in the thoracic walls and lungs. If the chest is capacious and the lungs are interposed between the chest-wall and heart, there may be no visible impulse. When, on the contrary, the parietes are thin and the organ is considerably enlarged, it produces visible shock which is exaggerated both in force and extent, while the apex-beat is displaced outward and in some instances downward, according to the degree of hypertrophy. In hypertrophy of the right ventricle there is apt to be visible pulsation in the epigastrium.

Palpation.—Confirmation of the facts perceived by the eye is obtained by the hand, and for the most part nothing more. In some instances careful palpation enables one to locate the position of the apex-beat when this is not visible, and to judge of the force and extent of cardiac contractions. In women with large mammary development in whom inspection and percussion are futile, palpation is often of great aid in estimating the size of the organ. The pulse of left-ventricle hypertrophy is full, strong, and inclined to be slow rather than accelerated. Increase in its rate comes on, as a rule, only when dilatation begins to gain ascendency and cardiac insufficiency to declare itself. It is a pulse of high and sustained tension, as shown by pressure of the finger on the artery and by Gaertner's tonometer or the sphygmograph. This is not due to stiffness of the vascular coats, but to the increased force with which the blood-wave is propelled by the powerfully contracting ventricle. Such a pulse is difficult to compress, but when the artery has been thus collapsed, the wave of blood is felt to strike the palpating finger strongly, while below the point of pressure the vessel is empty and its coats cannot be rolled beneath the finger, showing that they are not thickened and the increase of tension is not due to atheroma.

Percussion.—This forms our best and most reliable means of determining increase in the size of the heart. Absolute dulness may not be greater than normal, but the relative is, as shown by some one of the various modes of percussion described in the introductory chapter. Increase of deep-seated dulness to the left and upward is indicative of left-ventricle hypertrophy, provided, of course, the organ is not displaced.

The measurements given in the article on chronic myocarditis show that the distance between the midsternum and left nipple is not constant in all persons, but varies within considerable limits. It is not safe, therefore, to conclude that because relative dulness does not pass outside the nipple the heart is of normal size, yet, if dulness is found to extend beyond the vertical nipple-line, it is pretty sure evidence that hypertrophy exists. Likewise an increase of relative dulness to the right and downward betokens hypertrophy of the right ventricle. The measurements for the normal heart may be found in the introductory chapter.

Auscultation.—As one of the elements entering into the production of the first heart-sound is the muscular element, or that produced by the contraction of the wall and papillary muscles, the first sound is generally loud and booming. It is also apt to be rather prolonged. A more reliable criterion is obtained, however, by the study of the second sound at the base. Owing to the high pulse tension present in left-ventricle hypertrophy, the aortic second tone is accentuated, and this intensification is generally put down as one of the signs of hypertrophy. It is only of value, however, in connection with other signs. Similarly accentuation of the pulmonic second sound is an auscultatory sign of hypertrophy of the right ventricle. It should be remembered, however, that in children and young adults this tone is normally louder than the aortic. Auscultation is a much less reliable means of judging of the size of the heart than is percussion, since various conditions may temporarily alter the relative intensity of the sounds.

Diagnosis.—The recognition of cardiac hypertrophy depends not alone upon its degree, but also upon various conditions on the part of the lungs and thoracic parietes. Minor degrees may be assumed but cannot always be made out with certainty. On the other hand, a heart may be greatly hypertrophied and yet may escape our recognition because it is covered over by a voluminous or emphysematous lung, or the chest-wall may be so thick from fat and muscle as to render ordinary methods of diagnosis futile.

The clinical findings generally thought to warrant a diagnosis of cardiac hypertrophy are: (1) A full, tense pulse which is either slow or of normal rate, not accelerated; (2) a powerful, broad apex-beat which is displaced outward and perhaps downward; (3) increased cardiac dulness to the left and upward or, it may be, in all diameters; (4) a booming, low-pitched first sound and an accentuated aortic second sound. Hypertrophy of the right ventricle is shown by: (1) Epigastric pulsation, (2) increase of cardiac dulness to the right and downward, and (3) intensification of the pulmonic second tone. Should the condition of the lungs or thoracic walls not enable one to rely on the evidence furnished by the usual means of physical examination, then one may have recourse to the fluoroscope, which ought to settle the diagnosis beyond further question.

Differential Diagnosis.—It is hardly necessary to remind the reader that displacement of the heart towards either side may simulate hypertrophy, and therefore must be excluded. The most frequent source of error in this direction, however, lies in the fact that scoliosis, or that forward curvature of the spinal column, may cause the heart to lie close against the anterior chest-wall and to pulsate so forcibly and widely as to give an appearance of marked hypertrophy. In all cases, therefore, the shape and depth of the thorax ought to be carefully scrutinized.

In addition, I wish to refer to what Italian writers term "pseudo-cardiac hypertrophy," by which is meant a condition sometimes observed in young persons who are neurotic and have thin chest-walls with broad intercostal spaces. In such, owing to excitability, the action of the heart is apt to be rapid and unduly forcible. The apex of the organ strikes in the broad and thin intercostal space with what appears to be exaggerated force and abnormal breadth, or the entire cardiac area may heave strongly with each systole. The heart-sounds are intensified and ringing, and altogether the organ conveys the impression of abnormal strength. Consequently, unless the examiner makes careful measurements of deep-seated dulness, he is very liable to erroneously conclude that he has to do with a hypertrophied heart.

In all cases in which hypertrophy is suspected and physical signs are not convincing, a positive diagnosis of the affection should not be made until some condition has been discovered which is capable of inducing hypertrophy. In the absence of such predisposing conditions hypertrophy is not likely to occur, and hence one should be conservative in relying on physical signs if they are not very conclusive.

The liability to error was forcibly impressed upon me by the case of a young coloured man who was an applicant for life insurance and who was likely to be rejected by the examiner because of a supposed enlargement of the right ventricle. Absolute cardiac dulness was manifestly increased transversely, whereas relative dulness as shown by careful measurement was not augmented. It was then noted that he stood in a very faulty attitude with his shoulders thrown strongly backward and the scapulæ pressed closely against each other. In this position the spinal column was forced strongly forward to such an extent that it shortened the antero-posterior diameter of the thorax, and consequently pressed the heart against the anterior chest-wall so as to crowd the lung-borders aside and produce an apparent enlargement of the heart.

In some instances the diagnosis of cardiac hypertrophy is made almost at a glance, but in minor degrees its determination is only possible after one has carefully considered the degree of blood-pressure.

For the differential diagnosis of hypertrophy from dilatation the reader is referred to the succeeding chapter.

Prognosis.—The prognosis of the condition we are now considering may be said to be that of its cause. If this is of a progressive nature so that it is only a question of time when peripheral resistance will outstrip the accommodative ability of the heart, prognosis is ultimately unfavourable. It should be borne in mind, moreover, that the hypertrophied heart-muscle is likely to suffer degeneration; and when this sets in cardiac inadequacy is ultimately inevitable. If hypertrophy of the left ventricle is associated with a granular kidney and vascular changes, prognosis is influenced by the possibility of rupture of a brittle cerebral artery.

If hypertrophy has resulted from the abuse of athletic sports and the individual is still young, with healthy vessels, the condition may not prove serious providing the exciting cause is removed. In such persons, however, one should not ignore the possibility of congenital smallness of the arterial system.

Treatment.—Hypertrophy of the heart does not require therapeutic interference, and hence one should not attempt to lessen the vigour of cardiac contractions, as I have known attempted, by the use of aconite. The increased thickness of the heart-wall is a conservative measure. The aim should rather be to preserve hypertrophy and protect the heart from the inadequacy of overstrain. The underlying condition is the object of our solicitude. Valvular lesions, pericardial adhesions, chronic nephritis, congenital smallness of the aorta—these and many other causes cannot be removed. When detected, their existence should be stated to the patient and he should be warned against the danger of breaking down his hypertrophy by unwise physical efforts or any other injurious influences. If the cause lies in the excessive consumption of beer, in gluttony, in faulty athletic exercise, the patient should be plainly informed of his injurious practices and urged to desist before they lead to cardiac insufficiency.

If disordered action of the heart seems to call for digitalis, this remedy should be administered with great caution. As a matter of fact, such an invigorator of cardiac systole is not indicated unless signs of myocardial incompetence are actually present. When this is the case it is no longer the hypertrophy but it is the dilatation which calls for treatment. This will be found detailed in the appropriate place.

CHAPTER XXII

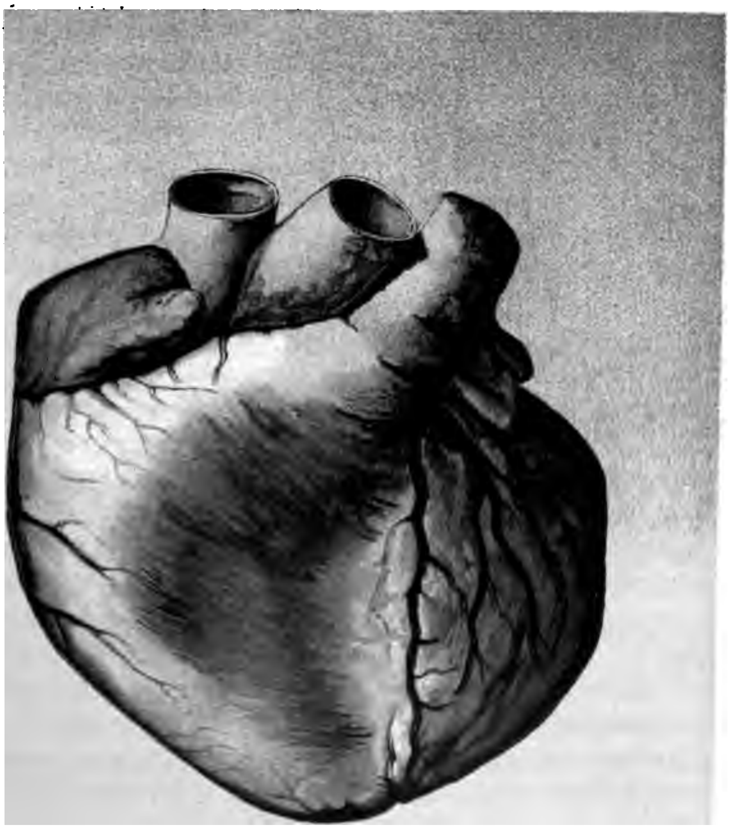
DILATATION OF THE HEART—RELATIVE MITRAL INSUFFICIENCY

I. DILATATION OF THE HEART

Except in cases of acute overdistention, dilatation of the heart is rarely primary, but secondary to some affection of an acute or chronic nature, as pericarditis, acute and chronic myocarditis, and valvular disease, and the diagnosis should not be made merely of dilatation, but, if possible, of the underlying pathological etiological condition. These have been already considered in previous chapters, and to them the reader is referred for particulars. More or less dilatation of the heart is recognisable whenever there is cardiac incompetence from whatever cause, but in this chapter the endeavour will be made to portray what may be considered as overstrain of the heart-walls, whether gradually or acutely induced, and independent of previous recognisable myocardial or endocardial disease.

Morbid Anatomy.—By dilatation of the heart is meant an increase in the capacity of its chambers due to rapid or gradual stretching of its walls. In most cases hypertrophy is combined with dilatation and has preceded the development of the latter. A dilated heart is as large or larger than one only hypertrophied, but the muscle is flabby, and the organ does not keep its shape when laid on the table. Extreme instances have been described in which the heart held up by the great vessels collapsed over the hand so as to cover it like a mushroom. This flabbiness is not characteristic of dilatation as such, but of all conditions of the myocardium in which the muscle has lost its tone and which have predisposed the organ to stretching. These are cloudy swelling, fatty degeneration, etc. Slight degrees of dilatation, as well as acute overstrain, are not attended by these myocardial changes—

PLATE III



N.B.Moore.

EXTERIOR OF HEART OF FIG. 42, SHOWING HYPERTROPHY AND DILATATION OF BOTH VENTRICLES.

they are often a part of the compensatory process attending valvular lesions. When, however, compensation breaks down and dilatation becomes extreme, the walls are found degenerated and flabby. The muscle is usually paler than normal and may be cloudy or of a brownish tint, due to the deposit of pigment.

As dilatation usually results from the further action of the causes that produce hypertrophy, it has much the same distribution. It may affect but one chamber or the heart as a whole. The trabeculæ and papillary muscles are naturally not concerned in the process of dilatation except in so far as the latter may stretch or lengthen in order to functionate properly within the enlarged chambers. Relative insufficiency of the valves is generally present and may be caused by stretching of their ostia or of the cardiac walls.

Etiology.—Cardiac dilatation may be said to be the result of a disproportion between the work the heart has to do and its ability to do it. This undue demand upon its energies may have existed for years in the form of prolonged high arterial tension, and only at length become disproportioned through degeneration and gradual waning of the heart-power. Not infrequently it is some unexpected call for extra effort that overpowers the heart, when without it the organ might have been able to perform its work successfully for years longer. It is in this way that dilatation so often succeeds compensatory hypertrophy in valvular disease. In many but not all such cases the integrity of the heart-muscle has been slowly undermined by the development of degenerative changes.

Such an exciting cause of dilatation may be a hasty run, a spurt on a bicycle, the lifting or carrying of a heavy weight, a prolonged debauch, etc. Anxiety, grief, and even fright, through their action on the inhibitory nerve of the heart, are capable of inducing a stretching of the cardiac walls through stasis in the cavities they inclose—the "*stauungs*" dilatation of the Germans.

Romberg lays stress on the deleterious influence in this respect of acute infectious diseases, and cites instances in which he has observed cardiac dilatation and ultimately fatal insufficiency follow an attack of influenza. I have notes of the case of a gentleman of fifty-seven whose dilatation and eventual death from progressive cardiac asthenia were attributable to his having carried a heavy travelling bag several blocks in Denver at an altitude of 5,280 feet. As this patient was moderately corpulent, weighing 192 pounds, his heart was probably not sound at the time of his exertion, and yet it had been adequate to all demands made on it previously.

We sometimes observe dilatation of the heart when we have no reason to assume that the organ is the seat of wide-spread structural disease and there may be no extensive post-mortem alterations that can be recognised. The heart-walls are soft and flabby, and that is all that can be said of them. In such a case the individual may have been anæmic, or his heart-muscle as well as his skeletal muscles has been weakened by years of close application to business or intellectual pursuits. Without any preparation such an individual takes to bicycling and at once indulges in long tours at a scorching pace up hill and down, over rough sandy roads and against heavy winds. Under the absurd impression that such exercise is good for his weak muscles, he heeds not his panting respirations and rapid heart-beats. But outraged Nature avenges the insult by permitting the gradual if not sudden development of cardiac dilatation.

I once had under treatment for a time a clergyman of middle age who had left-ventricle dilatation with relative mitral incompetence as the result of a single injudicious effort of this kind. As he expressed it, "he had pumped up" the steepest road on the banks of the Hudson River a thousand feet up the face of the Palisades—a road so steep that very few were ever able to make the ascent on their wheels. Although not aware of injury at the time, he not long thereafter began to suffer from alarming fainting spells, the first of which seized him while delivering a sermon. Fortunately for him his heart-muscle was sound and ultimately recovered its tone.

It is rather singular that I have been called on to treat for cardiac incompetence due to dilatation from strain three men whose occupations required them to inspect buildings in the process of construction. Failing to discover any satisfactory cause for their dilatation, I was led to inquire minutely concerning possible heart-strain, and found they were in the habit of climbing ladders or ascending many flights of stairs in order to reach the different parts of their buildings. It has seemed to me very reasonable to assume the likelihood of heart-strain by such exertion, and in the case of one of these men intermittence persisted in spite of treatment until he exchanged his work as building inspector for a sedentary occupation in an office.

In not a few instances the resistance of the myocardium is diminished by the inordinate use of tobacco or by sexual excesses, late hours and social dissipation, dancing, etc. In one case the young man breaks down in his nervous system, a second develops a cough, while still another manifests signs of cardiac dilatation that may have been suddenly or gradually induced. In most of such cases complete restoration of heart-power follows removal of the cause and appropriate treatment. In some the heart remains permanently impaired, and in others every fresh excess is followed by renewed dilatation until at length irreparable cardiac stretching and insufficiency remain. Such examples are not confined to the male sex.

Anæmia and chlorosis predispose to this form of heart-disease, and more than one society belle pays for the season's round of dancing and other gaiety by slowly or acutely induced incompetence of the dilated heart. Many a jaded matron who declares she is " worn out " by the demands of society is really suffering from serious though perhaps not extensive stretching of the heart-chambers. Her heart-muscle has grown flabby and is not always capable of entire restoration.

Fortunately the heart-muscle is susceptible of development the same as are the voluntary muscles. Were it not so, the athlete would be incapable of competing for the laurel wreath of victory. If, however, he is overtrained or if his training prove inadequate, the heart may be the part that suffers. Under such circumstances acute dilatation may result from a single contest. It usually affects the right ventricle, and stretching of the right auriculo-ventricular ring permits the " safety-valve action of the tricuspid " to come into play. (See the chapter on Tricuspid Regurgitation.) It is possible, however, for the left ventricle also to become acutely overdistended, as was shown by cases reported by Harold Williams.

If muscular incompetence of these valves is set up, then the strain is lifted somewhat from the walls of the left ventricle and shared by those of the left auricle and pulmonary veins. This is likewise a compensatory provision on the part of Nature, for without it there would be positive danger of diastolic arrest of the overtaxed left ventricle. In carefully trained athletes, or in the young with a robust myocardium, such a degree of cardiac strain is usually recovered from speedily and permanently. If the walls are not perfectly sound, or if there is sustained high blood-pressure in the aortic system because of vascular or renal disease, or if the heart is too frequently subjected to overstrain, permanent dilatation may result with all its consequences.

Finally I desire here to dwell on the harm in this regard that is likely to accrue to young children from the immoderate use of the *bicycle* and from games that necessitate long, hard running. There is *actual, not fancied,* danger of cardiac dilatation. If their hearts are sound no permanent injury may ensue, unless, of course, the strain be too oft repeated. In many cases the children have suffered from undetected rheumatism and latent pericarditis or endocarditis, and in such, unrestrained indulgence will surely result disastrously through the development of dilatation of the heart with possible coincident inflammation of some of its structures.

The heart of a growing boy endures a degree of strain disastrous in a man of forty, and yet if overstrain be too frequently repeated during one of the "fatigue periods" of childhood, it may, I am convinced, ultimately enfeeble the heart's resistance even in a boy. The overdistention of the heart is due probably to ineffectual systoles, which allow a residue of blood to remain in the distended cavities, while with the continuance of muscular effort and deepened respirations blood is passed on to the heart more rapidly and in larger amounts than can be expelled.

Another factor that should not be ignored in considering the question of cardiac strain are those unknown "fatigue products" developed during severe exercise and to which particular attention has been called by Clifford Allbutt.

Symptoms.—The symptoms of cardiac dilatation develop slowly or rapidly according to the development of the dilatation. The stretching and often thinning of the heart-walls impair their contractility, and there is a tendency to stasis within the organ which leads to loss of equilibrium between the arterial and venous streams. As the heart-power begins to fail the pulse grows more rapid, often irregular in force and volume, and in many cases intermittent. Cardiac impulse becomes weaker, its area of dulness increased, and its sounds feeble.

The patient begins to notice more or less breathlessness on exertion and a feeling of unwonted lassitude that may amount to actual weakness. His colour changes from the reddish hue of health to the bluish gray tint of increasing capillary stasis. As cardiac inadequacy advances, symptoms of visceral congestion appear. The urine lessens in amount and becomes of high specific gravity, often containing a trace of albumin. The liver increases in size, which may cause a feeling of fulness in the right hypochondrium or of dull pain in the back below the shoulder. Its inferior margin becomes more or less distinctly palpable, being smooth, firm, and rounded, and palpation of the organ may be somewhat painful.

Congestion within the gastro-intestinal veins is shown by impairment of appetite and more or less flatulent indigestion, so that the patient feels bloated after meals and complains of windy constipated bowel movement. Piles may develop, sexual power become deficient, and women are apt to suffer from leucorrhœa and derangement of menstrual function. Congestion within the lower extremities leads at first to puffiness of the ankles, which by night feel tense and uncomfortable. When the shoes are removed the skin is found creased, and a ridge indicates where the upper edge of the shoe pressed. After a night's rest this swelling of the feet and ankles may have subsided, but as cardiac incompetence progresses, œdema remains permanent. Pitting on pressure is now pronounced and found to gradually extend upward.

Thus gradually but steadily grow the symptoms of failing circulation, and at length, if the condition is not arrested by treatment, the patient is compelled to keep his room. Pulmonary congestion is no longer shown merely by dyspnœa on effort, but by cough with frothy, perhaps bloody expectoration, and by orthopnœa. Talking causes breathlessness and so much fatigue that the patient dreads or even shuns the effort. Examination of the lungs discloses more or less dulness at the bases behind with fine crackling râles—in short, the signs of hypostatic congestion.

The apex-beat is now imperceptible or is but a feeble tap much outside the left nipple. Absolute and relative cardiac dulness are greatly increased, of a quadrangular outline, and in extreme cases may extend from close to the right nipple on the one hand nearly to the anterior axillary line on the other.

The heart-sounds are almost inaudible and there is very apt to be a systolic murmur denoting relative mitral or tricuspid insufficiency or both. The leak through the tricuspid valves is shown by the positive venous pulse in the external jugulars and liver. The veins of the neck stand out like blue cords, and if pulsation in them is not apparent at all times, becomes plainly visible so soon as the patient coughs or makes a forcible expiratory effort. The pulse is now rapid, feeble, and often arrhythmic.

The state of things has now become pitiable, and if not relieved grows steadily worse, with the development of general dropsy, transudation into the serous cavities, somnolence, even low muttering delirium and death. This last may occupy hours, appearing to come from gradual exhaustion, or it may be ushered in by pulmonary œdema. In such a case fine crackling râles develop, spread throughout the lungs, and at last become plainly heard at a distance with every laboured respiration. In other instances pulmonary infarction occurs, as shown by cough and the expectoration of bright-red blood. In some the heart stops abruptly and unexpectedly, while the patient is at rest or making some slight exertion, although this sudden cessation of the heart's action has been preceded for several days by signs of such increasing weakness that its final arrest is scarcely a matter for surprise.

In some cases symptoms of cardiac dilatation, even extreme, persist for many months or even two or three years. I recall a man of fifty odd whom I treated in 1893 and who was a striking example of this chronicity. For more than a year he had been incapacitated for attention to business and yet had managed to keep about in spite of very apparent shortness of breath, a rapid, exceedingly arrhythmic pulse, enormously increased cardiac dulness, and feeble heart-sounds. The liver could be felt thin-bordered and hard, and this patient eventually died apparently from the pressure-effects of ascites rather than from independent asystolism. This man's heart was undoubtedly degenerated and very thin-walled. It might be reckoned as an example of cardiac inadequacy from chronic myocarditis, but it was a typical picture of chronic dilatation of the heart.

I have recently treated with highly gratifying results, by means of baths and resistance exercises, a powerfully built man of thirty-eight who was suffering from the effects of heart-strain four years before. When in apparently perfect health he endured a day of terrible fatigue from the exertion of journeying in a severe snow-storm at the altitude of 18,000 feet. By night he was completely exhausted, looked blue, felt cold, had a feeling of great præcordial oppression, and could not get his breath. After a few days' rest he felt better and returned to the East. He remained at business, but when summer came on, went abroad for a rest, and in London consulted a well-known medical authority. By him he was told he had suffered a heart-strain and was advised to give up business. He did not do so, however, with the result that he gradually developed symptoms of chronic heart-disease. For some months before I saw him he suffered from dyspnœa of effort, a feeling by night of profound exhaustion, and the conviction that he was liable to die suddenly at any time. He nevertheless remained at business.

When I first saw him he presented the signs of mitral regurgitation with secondary enlargement of the heart, chiefly of the left ventricle. Lungs were negative, but the liver was palpable. There was no pitting, but the tissues everywhere felt tense and hard, and the patient said he "felt swollen." Preliminary treatment by rest in bed, cathartics, and a milk diet for two days reduced capillary stasis, improved the quality of the pulse, and removed the patient's sense of air-hunger. There was 2 per cent of albumin in the urine, but although repeated search was made for casts, they were never found.

At the end of less than four months this patient declared he felt perfectly well and desired to return to business. The heart was manifestly smaller and its action greatly improved, but the mitral systolic murmur still remained. It was less loud and less harsh, however, and the first sound, originally inaudible, could be heard distinctly. The liver could not be felt, but albuminuria persisted. This man had never had articular rheumatism or any disease to lead to endocarditis, and prior to his arduous mountain climbing had never had even the slightest symptoms of heart weakness. I have no doubt that his heart-muscle has suffered in its integrity, but I look upon this as an instance of chronic left-ventricle dilatation due primarily to strain and leading to relative mitral incompetence.

Acute dilatation of the heart from strain most commonly affects the right ventricle, but it may also take place in the left. This was shown in the cases of the young men who were examined by Harold Williams immediately before and after a run of twenty-five miles. Cardiac dilatation was shown by exhaustion, cyanosis, a rapid thready pulse, manifest increase in heart's dulness both to right and left, and by a systolic murmur having the characters of a mitral bruit. The safety-valve action of tricuspid regurgitation may be rather quickly induced as compared to the time it would take to set up such a left-ventricle dilatation as would produce relative mitral incompetence. In a run of twenty-five miles requiring three or four hours, time would be given for the safety-valve action of mitral insufficiency to occur. Were this not so, the left ventricle would be subjected to a degree of strain that might prove dangerous and even fatal.

The symptoms of acute heart-strain are those of deficient arterial circulation (relative arterial anæmia), a rapid, weak, and it may be irregular or intermittent pulse with signs of an overloaded venous and pulmonary system, dyspnœa, exhaustion, præcordial discomfort, perhaps pain, and cyanosis, congestion of the liver, and positive venous pulse of tricuspid regurgitation.

Within the last year I have seen two instances of acutely induced dilatation of the right ventricle in powerfully built young men belonging to college foot-ball teams. One of them walked off the field after the contest was over, but he looked blue in the face, complained of dull pain behind the lower end of the sternum, and the physician having charge of the team detected increase of absolute dulness to the right. Symptoms did not disappear for a number of days, and it was months before the heart's action regained its wonted steadiness.

When the heart-muscle is healthy, acute dilatation from overstrain may be recovered from, yet if too frequently repeated may without doubt result in permanent cardiac incompetence. I believe this is a very positive danger attending college athletics, particularly in foot-ball and rowing matches.

When the heart-muscle is not healthy, and therefore when persons have passed their forty-fifth year and have arrived at a time of life in which the state of the myocardium is questionable, acute cardiac dilatation from overstrain becomes a very serious matter. The first symptoms of heart-strain may be recovered from, but more or less inadequacy is likely to remain. In time, as a result of renewed but less severe strain, evidence of weakness sets in and the patient ultimately presents a clinical picture of gradually increasing dilatation of the heart.

Physical Signs.—*Inspection.*—The multiplicity of conditions which affect the results of inspection renders this means of investigation of comparatively small value. The eye may perceive the well-known manifestations of cardiac weakness, but it cannot furnish information as to the actual state of the heart. There is usually an absence of visible cardiac impulse, but this alone is of no value, since it is normal to many individuals to have no visible apex-beat on account of the volume of the lungs or the thickness of the chest-wall. In emphysema, which so commonly leads to ultimate dilatation of the right heart, visible impulse is also likely to be wanting. In all cases in which the apex-beat is not plainly visible the patient should be placed in a strong light and inspection made across the front of the chest from the side or from above downward. If dilatation exists a feeble apex-shock may thus be sometimes perceived outside of and below the nipple or in the epigastric notch.

Palpation.—Aside from the knowledge which this affords concerning the pulse and hepatic congestion, palpation is of service in the estimation of the feebleness or strength of cardiac contractions. Thin-walled and dilated hearts may give no perceptible shock to the chest-wall, or they may occasionally produce a sudden quick tap whenever the organ gathers itself, as it were, for an extra effort. When the apex-beat persists, it is not like the broad heaving impulse of hypertrophy, but is a circumscribed feeble stroke of a slapping character. This is particularly the case in long-standing dilatation with still some degree of efficiency.

Percussion.—This is, as a rule, our most valuable means of determining if dilatation of the heart is present, but it may afford very unreliable evidence in cases of pulmonary emphysema. If the state of the lungs and of the thoracic parietes renders percussion available and if dilatation exists, the area of deep-seated if not of superficial dulness is found increased in accordance with the degree of dilatation and the chambers affected. In dilatation of the left ventricle relative dulness is increased towards the left and upward, while in stretching of the right heart it is augmented to the right and downward. When general dilatation of the heart exists the area of dulness is found to have assumed a quadrangular outline with broadly rounded corners and sides.

Auscultation.—The heart-sounds are feeble, altered in relative intensity and rhythm, and are frequently accompanied or obscured by murmurs of relative mitral or tricuspid incompetence. The systolic tone becomes short and valvular like the second, and as the diastolic pause is shortened the rhythm of the sound tends to disappear, the tones following each other in quick succession something like the ticking of a small clock or watch. In cases of extreme weakness and rapidity the ear may be able to detect but one sound. The aortic second tone is usually diminished while the pulmonic second is accentuated. If murmurs exist they are, as already stated, due in most cases to relative incompetence of one or both auriculo-ventricular valves. The auscultator should not forget, however, that it is quite possible for bruits of pre-existing valvular disease to be present, and therefore he should be wary about concluding that the murmur is necessarily a relative one. In many cases he must await the result of treatment before deciding definitely on its real nature.

Diagnosis.—The recognition of cardiac dilatation is ordinarily not difficult, especially if it has been acutely induced or has progressed to the production of considerable inadequacy. Minor degrees of stretching are not always easy of detection and require minute inquiry into the history and symptoms, as well as painstaking physical examination. In slowly induced dilatation there is history of gradual onset and progressive increase of symptoms of cardiac incompetence, while there are the clinical findings of (1) a more or less rapid and feeble, it may be irregular or intermittent pulse; (2) feeble, tapping, or even imperceptible cardiac impulse; (3) increase of relative and perhaps superficial dulness in one or more directions according to the chambers affected; (4) feeble ticking sounds and perhaps systolic murmurs of relative mitral or tricuspid insufficiency.

In acute overstrain the phenomena of cardiac embarrassment follow some unusual exertion and are easily recognised, while the patient is apt to display more or less cyanosis and other evidence of impending stasis.

Differential Diagnosis.—Acute dilatation of the heart can scarcely be mistaken or confounded with any other condition provided due attention is paid to history and objective symptoms. Distention of the cardiac cavities that has developed more slowly and has grown extreme may, however, be mistaken for pericardial effusion. The differential points are fully dealt with in that chapter, but special emphasis may here be laid on the necessity of determining the relation of the outer and inferior margin of deep-seated dulness to the position of the apex-beat. In cardiac dilatation dulness does not pass beyond the limits of cardiac impulse, whereas in pericardial distention the apex-impulse is situated within the outer border of dulness. This, and this alone, is the trustworthy criterion of difference between the two affections. In other respects there is often a striking similarity.

Dilatation and hypertrophy can scarcely be confounded if due attention is paid to the characters of the pulse, to the nature of the impulse, and to the greater feebleness and rapidity of the sounds in dilatation. A simply dilated and yet not specially degenerated heart cannot often be distinguished from a degenerated and hence secondarily dilated organ, and it is not always prudent to attempt such distinction.

Prognosis.—Dilatation of the heart should never be regarded as a trivial matter, and yet the degree of its gravity depends upon the state of the heart-muscle, the extent of the dilatation, and the length of time it has existed. It is the integrity of the myocardium in the young and in trained athletes which in them makes the heart recover so quickly and well from the overdistention caused by strain. On the other hand, it is the likelihood of the heart-walls being not quite sound which renders prognosis serious when dilatation supplants hypertrophy or when elderly individuals suffer heart-strain. Stiffened arteries do not necessarily mean that the heart-walls are seriously degenerated, and experience abundantly proves that in some cases proper treatment may restore a dilated heart even when the vascular coats are thickened. Nevertheless, under such conditions stretching of the cardiac chambers is always a grave affair, and only too often there is but little prospect of the heart-walls again becoming as sound as before the injury.

Cardiac dilatation associated with a granular kidney may always be said to furnish a very grave prognosis, for the resistance in the arterial system is so great that in all likelihood the heart has been gradually yielding, and having once given way, cannot regain its lost adequacy. In such a case the prognosis depends both upon the state of the heart-muscle and the possibility of lessening the high pulse-tension by treatment.

In the face of both arteriosclerosis and interstitial nephritis a seriously dilated heart is likely never again to recover its former compensatory hypertrophy.

It is so evident a fact as to scarcely require statement that the more extensive the dilatation the more serious the prognosis. With care and proper management minor degrees of the condition may endure for a long time—even years—yet in such a case the tenure of life is always uncertain, for the reason that some additional and unexpected strain may at any time convert a not altogether unfavourable into a most serious prognosis.

Stretching of the auricles or of the right ventricle is not so grave a matter as is left-ventricle dilatation. Owing to the thinness of their walls they may not be so amenable to treatment—that is, not so likely to have their hypertrophy restored; but on the other hand, the right ventricle is more likely to be relieved by stretching of its auriculo-ventricular ring, and therefore is less liable to paralytic overdistention and diastolic arrest. This consideration leads me to the belief that in cases of left-ventricle dilatation the development of a systolic murmur at the apex is a favourable rather than an unfavourable prognostic indication. Such a murmur is usually held to indicate relative mitral insufficiency, and by the giving way of the mitral valve a part of the excessive endocardial blood-pressure becomes transferred to the auricle and pulmonary veins, and thus the wall of the left ventricle is relieved, in a measure at least. I can recall more than one instance of sudden and unexpected death in men whose hearts were seriously dilated and in whom such a systolic apex-murmur did not exist. On the other hand, I have more than once observed that when the mitral valve gave way the progress to asystolism was gradual, and death was usually preceded by the symptoms and signs of venous stasis, extending through a period of weeks or months.

It is also evident that prognosis is largely governed by the length of time the dilatation has existed. If the myocardium is still healthy, as in the young, a chronic dilatation may yet be recovered from. In persons past middle age the condition is likely to resist treatment if it has become chronic, and if in such the pulse is persistently arrhythmic, it is to be looked upon as an indication that the heart-muscle is not sound or that the auricles are greatly dilated. According to Radizewsky, habitual irregularity of the pulse points to preponderating degeneration of the walls of one or both auricles. If this be correct, then arrhythmia in connection with chronic dilatation of the heart is a better prognostic sign as regards length of life than is tachycardia with perfect regularity of rhythm due to a dilated ventricle. In acute dilatation from overstrain, as in foot-ball or mountain climbing, the prognosis is as a rule good, for with rest and proper treatment the heart is likely to return to its former healthy condition. Repetitions of its abuse may, however, eventually induce permanent inadequacy.

Another element that enters into the question of prognosis is the degree of subjective symptoms produced by the dilatation. If dyspnœa be less than one would be led to expect from the apparent gravity of the condition, the case is likely to pursue a chronic course; if, on the other hand, the shortness of breath be out of proportion to the apparent size of the organ, or to the amount of exertion performed by the patient, or if the dyspnœa assumes the form of cardiac asthma, then the prognosis is bad, unless the urgency of this symptom can be accounted for by an associated emphysema or bronchitis.

If skilful treatment succeeds in producing only temporary improvement, and the heart drops back to its former state so soon as treatment is discontinued, or less vigorous, it is an indication that the heart-muscle is either too weak to be regenerated, or the peripheral resistance is too great to be permanently overcome. A steady though gradual loss of ground, in spite of treatment, proves that very little is to be expected from any management, no matter how skilful.

Except in cardiac strain of effort in the young, one should never venture to prognosticate the length of time it will take for the heart to be restored to health, or in those in which this is manifestly impossible, to predict how long the disease is likely to last. If the left heart is the one chiefly affected, the end may come suddenly and unexpectedly, but in those cases in which the right heart is the one chiefly at fault, and particularly when the right auricle is much dilated, pulmonary infarcts are very likely to occur and to be the immediately determining cause of death.

Two states of mind on the part of the patient, if they come on after the disease has existed for some time, and serious symptoms of stasis are present, always make me apprehensive of the near approach of the end. These are, great restlessness and an ill-defined nervousness that keep the patient constantly moving about, and on the other hand a sudden lull in the patient's sense of distress. After days or perhaps weeks of severe suffering he suddenly has a day in which he feels remarkably well and free from distress. This is often like the calm that precedes the storm. I have more than once seen death speedily succeed such a day of apparent well-being. Apropos of the former state I recall a large middle-aged man presenting all the symptoms and signs of extreme cardiac dilatation whom I examined in consultation with Dr. Harrison late one afternoon and in whom I saw no evidence that the end was imminent; he had been in that condition for a week, and, although very dyspnœic, walked into an adjoining room. He displayed, however, an indescribable restlessness, and less than three hours after we left him he died suddenly.

Treatment.—In dilatation of the heart suddenly induced through strain the first indication is to place the patient at rest in the recumbent or semi-recumbent position for the purpose of lessening the heart's work so far as that is possible. Muscular inaction and a more tranquil respiration bring the venous blood to the right heart less rapidly, and if the circulatory apparatus is healthy, as in the youthful athlete, Nature alone, under favourable conditions, will speedily effect a restoration of the blood-stream to its former equilibrium.

If, on the contrary, the patient's age or state of general nutrition justifies the inference that the heart cannot return to normal without further aid, then a mercurial pill and digitalis should be prescribed. By unloading the portal system the cathartic tends to restore balance within the abdominal vessels and secondarily in the system at large. Five grains of blue mass followed by ½ ounce of Epsom salts eight hours thereafter, or a grain or two of calomel at bedtime and a glassful of the solution of citrate of magnesia next morning, or any one of the numerous aperient waters in the market, will prove highly efficient to that end.

The purpose of the digitalis is to slow down the heart and enable it to empty its distended cavities effectively. This may be accomplished by the administration of 10 to 15 minims of the tincture every six hours for four or five days. Improvement will be shown by a reduction in the rate and corresponding gain in the force and strength of the pulse, by disappearance of cyanosis and other signs of venous congestion, by increased diuresis, and a gradual return to normal in the size and sounds of the heart. Strychnine and nitroglycerin will probably not be required. It is well, however, to insist on light yet nutritious diet until the normal state of the circulation has been regained.

When *permanent cardiac insufficiency* is threatened from repeated heart-strain or from the gradual giving way of hypertrophy in the face of relatively too great peripheral resistance, the principles of treatment must be the same as in any other form of heart-weakness. The first indication is to relieve the overtaxed heart. Therefore the patient must be put at physical rest for a length of time which is to be determined by results. To the patient's query, "How long must I stay in bed?" do not permit yourself to make a definite answer, but tell him that is to be determined by the rapidity and degree of improvement. In other respects the same general plan of action previously detailed for cases of lost compensation is to be followed, but varied to meet the peculiarities of each case.

Most cases of cardiac dilatation which the physician is called on to treat are not instances of acute strain, but of chronic cardiac insufficiency. They are to be managed, therefore, in accordance with the principles laid down for the treatment of inadequacy from chronic myocarditis, and the reader is referred to that chapter for details.

There are three measures, however, of which it may be well to speak with special relation to cardiac dilatation:

(1) *Bloodletting.*—Occasionally a patient is encountered who from one cause or another is suffering from great overdistention of

the right heart. The action is extremely feeble and disordered, the face is congested, the veins are turgid, dyspnœa is profound, the lungs are filled with râles of pulmonary œdema, there is cough and bloody, frothy expectoration, there is no œdema, but the extremities are cold and cyanosed. Percussion shows enormous dilatation of the heart, and, so well as can be determined, the heart-tones are very weak and impure. Dissolution appears imminent. Under such circumstances the physician realizes that whatever is to bring relief must be done quickly.

There is not time for digitalis and cathartics to be tried; they are too slow. In such an extremity there is nothing that usually affords such prompt relief as venesection. Twenty or more ounces of blood taken from the arm are generally followed by diminution of cyanosis, noticeable improvement in the quality of the pulse, and increased clearness and strength of the heart-sounds. Of course such improvement will be only temporary if nothing else is done. This treatment must be followed up, therefore, by the judicious use of digitalis, strychnine, and cathartics, to secure what is gained by the abstraction of blood, since this latter is of course only a temporary measure resorted to for the dire emergency. Many a patient's life has been saved by such treatment, but, unfortunately, it will not save all.

(2) *Nauheim Baths.*—By some authors who seem to make a fad of this mode of treatment these saline and effervescing baths are advised even in cases of extreme and long-standing dilatation. Judging from my experience, such treatment is a mistake in cases in which, from the arrhythmic pulse, enormous area of cardiac dulness, and feeble tumultuous heart-sounds, it is clear that the cardiac walls are too thin to ever regain their old-time power. Theoretically these baths should unload the cardiac cavities and thus assist the heart in its labours. As a matter of fact, I believe in these cases they do not accomplish this result. Such persons cannot be materially benefited by anything, yet if any remedy can help them it should be digitalis, strychnine, and cathartics judiciously administered for an indefinite time.

(3) *Resistance Exercises.*—If given by an attendant who thoroughly understands how to apply the proper degree of resistance to a feeble heart, these exercises may prove of real benefit even to a seriously dilated heart. They are supposed to relieve

the cardiac cavities by diverting a part of their contents to the peripheral vessels, and as a matter of fact they are followed by a demonstrable decrease in the area of cardiac dulness. Nevertheless I do not believe it is possible to create so remarkable a decrease as is claimed by Theodor Schott, who finds in this effect a means of differential diagnosis between a dilated organ and a pericardial effusion. When employed in the condition now considered they must be given with very great gentleness, and movements are to be omitted which necessitate the elevation of the arms above the head as well as bending of the trunk at the hips, which are capable of augmenting dyspnœa and aggravating the dilatation. Even when permanent improvement in the patient's condition does not result it is generally found that for a time at least there is a lessening of his distress and an improvement in his colour. I therefore recommend their trial in all cases of chronic cardiac dilatation.

Should cases of chronic cardiac incompetence be not improved by the measures just spoken of, then it is reasonable to infer that the case has passed beyond the stage in which anything more is to be hoped from treatment than the amelioration of symptoms. One may yet do what he can with digitalis, strychnine, nitroglycerin, diffusible stimulants, and cathartics. In the majority of cases morphine will now have to be given, and if administered hypodermically to secure comfortable nights, the remedy is generally of the greatest service. In many instances morphine thus given will prolong life and ease the sufferer's path to the grave.

Only a few months ago Dr. George F. Roberts, of Minneapolis, called me to see a gentleman of nearly sixty who presented a typical picture of a dilated heart. The myocardium was probably degenerated, but his arteries were soft and urine was negative, so that one could not say there was more than cardiac incompetence from dilatation. He was in bed and dyspnœic, but his suffering arose from vertigo, which came every few minutes and lasted from a few seconds to a minute or more. During the vertigo his radial pulse wholly disappeared and the heart-sounds became exceedingly rapid and feeble but perfectly regular; the cavities were not being emptied. Suddenly the action of the heart would change, becoming slow, strong, but irregular; the pulse would return and the patient would exclaim, "There! it is gone!" meaning, of course, his dizziness.

The cervical veins were distended, liver was palpable, urine scanty, but no œdema and no turgescence of the superficial veins over the trunk and limbs. Cardiac impulse was absent and the area of dulness enormously increased and of a quadrangular outline. It seemed as if the large vessels of the abdominal and portal systems were holding the most of the blood.

Venesection was indicated and might have afforded temporary relief, but for certain reasons it was not performed. Instead, nitroglycerin, camphor, and valerianate of caffeine were injected subcutaneously at short intervals and the bowels were opened by calomel and a saline. Vertigo was relieved by this means, but circulation was not materially improved. The heart-walls were too greatly stretched and probably degenerated to regain their adequacy, and the patient died about ten days subsequently.

II. RELATIVE MITRAL INSUFFICIENCY

This condition is the result of such a stretching of the left auriculo-ventricular ostium that the mitral valves are incapable of adequately closing it during ventricular systole, and more or less regurgitation into the auricle is permitted. It is what Balfour terms curable mitral regurgitation. It is sometimes spoken of as functional mitral incompetence.

Pathology.—This is really that of dilatation of the left ventricle, and as such has been already described, besides having been repeatedly mentioned as a secondary manifestation of aortic-valve disease. The condition may be acute and transient or chronic and permanent. In the former class there may be no changes discovered at the necropsy further than simple dilatation of the left heart and mitral ring. Or there may be found disease of the aortic valves and orifice which with its myocardial changes has led to dilatation of the mitral ostium. In the chronic form the heart-muscle is generally degenerated, the papillary muscles elongated, flattened, and sclerotic, especially at their tips, and the mitral leaflets themselves may be longer and broader than natural, showing Nature's attempt to render them competent.

Etiology.—The etiology of relative mitral insufficiency includes (1) *predisposing* and (2) exciting causes. Among the former are to be reckoned all diseases of the myocardium, papillary muscles, and aortic valves which lead eventually to overdistention of the left ventricle and auriculo-ventricular ring, or to incompetence of the mitral valves without stretching of the heart-wall. Under this latter head are included those rare cases in which a mitral leaflet has been allowed to bulge backward into the auricle in consequence of rupture of a tendinous cord or of stretching of a papillary muscle.

The exciting causes are all influences which subject the left ventricle to overstrain. In reality the ventricle is subjected to overstrain when it dilates in the last stage of aortic incompetence or aortic stenosis and the mitral valve gives way, but in the class of cases now referred to there is some enormous physical exertion, and endocardial blood-pressure reaches so high a degree as to cause overdilatation of the ventricle. Efforts capable of this are boat-racing, prolonged running, etc. That such a result actually occurs even in healthy young men was proved beyond doubt by the observations of Harold Williams, who found that in 13 men who came in exhausted and done up after a run of twenty-five miles there were plain signs of left-ventricle dilatation with a blowing systolic apex-murmur in 11. Although I have not seen statements to that effect, I have often wondered if this were not the condition in amateur oarsmen who sometimes faint away at the close of a contest in which they have been forced to row to their utmost for miles.

In many cases there is doubtless a double cause at work—i. e., a predisposing and an exciting one. Thus in the chapter on Chronic Myocarditis is narrated an instance of a man who had been treated for mitral regurgitation supposedly of endocarditic origin. The necropsy, however, revealed not a valvulitis, but an astonishing degree of myocardial degeneration and a congenitally small aorta. Moreover, there was a history of acute heart-strain —so that in this case there were several factors responsible for the relative mitral incompetence. Whether the abnormally small aorta had led to the myocarditis or whether this latter had been caused by some infection, as articular rheumatism, of which there had also been a history, the degeneration of heart-muscle rendered the left ventricle incapable of successfully coping with the long-continued strain of the aortic condition. Hence, given this twofold influence, it only required the third element of acute strain to bring on the relative mitral insufficiency from which the patient never recovered.

This form of mitral regurgitation is occasionally seen in anæmic girls, or at all events in such there is frequently detected a systolic murmur loudest in the mitral or pulmonic area, or in both. The presence of such murmur, together with signs of cardiac hypertrophy and symptoms of inadequacy—i. e., breathlessness on exertion—warrants the conclusion that the valves are relatively incompetent. It is to this class of cases in particular that Balfour applies the term curable mitral regurgitation. There has been much dispute as to whether there is actual valvular incompetence, or the systolic murmur is not in reality only an accidental (hæmic) one. Granting, for the sake of argument, that regurgitation actually occurs, and I believe it does in many cases, we must regard the anæmia as a predisposing, and some other factor, perhaps exertion, as an exciting cause of the mitral leak.

So manifold are the conditions which can lead to this form of regurgitation that to enumerate them all is practically impossible. It is consequently only necessary to reiterate that the exciting cause is undue strain either slowly or suddenly induced and acting either alone or in conjunction with some antecedent influence.

Symptoms.—These are the same as in mitral reflux of endocarditic origin and need no description. They may, however, be rapidly or slowly induced according to the nature of the cause.

In curable mitral regurgitation of chlorosis and anæmia visceral congestion is rarely pronounced, although there may be slight ankle œdema. This is probably attributable to the blood-state even more than to circulatory disturbance. The chief symptom in such cases is shortness of breath, and this may also be due in large part to deficient oxygen-carrying power of the blood, since it is often present in females who have no regurgitation. Other symptoms, as weakness, anorexia, digestive disorders, and constipation, belong to the anæmia. The same may be said of scanty menstruation or amenorrhœa, since women with organic mitral incompetence are apt to display a tendency to menorrhagia.

Physical Signs and Diagnosis.—There may be said to be nothing in the physical findings distinctive of relative as contrasted with organic mitral regurgitation, and hence they do not call for recapitulation. The chief difficulty in diagnosis lies in cases of anæmia and chlorosis—i. e., in determining whether or not the systolic murmur is accidental. The reader will find details on this point in the introductory chapter.

The following differential points are, however, of aid: (1) A history or not of articular rheumatism or other cause of endocarditis. (2) The recognition of the blood-state. (3) Organic mitral murmurs are usually transmitted to the back while accidental ones are not. (4) A chlorotic systolic murmur is generally most distinct in the pulmonic area, yet is audible in other regions. (5) Secondary heart-changes are apt to be more pronounced in the organic form, particularly if the patient is conscious of symptoms. A palpable liver speaks for organic mitral regurgitation.

There are unquestionably many cases in which the differential diagnosis must be left to the results of treatment.

Prognosis.—This depends upon the cause of the insufficiency. If it is secondary to aortic-valve defects or to myocardial degeneration, prognosis is bad, both in regard to the mitral leak and to the effect of this on the primary disease. When due to acute heart-strain in the young, prognosis is usually good, since the regurgitation disappears after appropriate management. In the middle-aged and old, on the contrary, there is small likelihood of the insufficiency being wholly repaired. Prognosis is favourable also in the anæmic, since it disappears after the state of the blood has been restored to normal.

Treatment.—The treatment of relative mitral incompetence is both that of the cause and of the cardiac condition. When it has arisen from strain the organ must be put at rest as the prerequisite to success of further therapeutic management. In this as well as all other respects the treatment is the same as in dilatation of the heart just considered and in mitral regurgitation of endocarditic origin.

Anæmia and chlorosis are to be combated by iron, arsenic, and foods rich in nucleo-albumins. For this class of patients life in the open air is most important, and hence it is well to send such to some seaside resort, to a spa the waters of which are impregnated with iron, or to the climate of the mountains which, according to recent observations, appears to favourably influence such blood-states.

In relative mitral insufficiency digitalis is of pronounced utility and should be prescribed in connection with general tonics. Highly gratifying results also follow the Nauheim baths and resistance exercises. Some of the most brilliant recoveries I have ever observed have been in precisely this class of cases.

CHAPTER XXIII

FATTY HEART

CARDIAC INADEQUACY OF THE CORPULENT

Fatty heart is the term most commonly employed to designate, not fatty degeneration of the heart-muscle, but a deposit of fat beneath the epicardium and between the muscle-fibres—a condition variously styled fatty overgrowth and fatty infiltration.

Morbid Anatomy.—In this disease the subepicardial layer of adipose tissue is strikingly, sometimes enormously, increased. The fat is particularly abundant in the interventricular and interauricular grooves, especially the latter, and along the branches of the coronary arteries. It is usually thicker over the right than over the left ventricle. It is not only deposited on the surface of the organ, but makes its way between the bundles of muscle-fibres, which, examined microscopically, are seen to be more or less widely separated and to have become attenuated or atrophied. In some instances there may even be masses of fat beneath the endocardium.

Pathology.—It is generally believed that when cardiac insufficiency declares itself in fat people it is owing to an excessive deposit of adipose tissue upon the heart or to fatty degeneration of the heart-muscle. Romberg has, however, set forth in so admirable a manner the real pathology of this condition that I shall avail myself of much of what he says.

He agrees with Leyden in the view that the old conception of fatty heart as an independent affection must be abandoned, and, instead, makes the term fatty heart include those disturbances of heart action manifested by the obese which either bear a direct relation to their obesity or have developed independently. That their cardiac insufficiency is not due to fatty overgrowth is substantiated by the observation that hearts loaded down with adipose tissue have not always given signs of inadequacy, and on the other hand that such as were manifestly insufficient during life have not always shown a deposit of fat sufficient to account for the weakness.

The cause of the heart difficulty resides, therefore, in some other condition, and this Romberg finds to be relative smallness and weakness of the heart-muscle—i. e., disproportionate to the demands made upon it by the condition of general corpulence. In some fat but muscular individuals the heart is correspondingly large and muscular, and symptoms of cardiac inadequacy do not appear. Other corpulent individuals of indolent habits are anæmic and have a flabby musculature. In them the heart-muscle, rendered weak and flabby through anæmia and want of exercise, is incapable of responding adequately to the work required of it, by the great exertion of moving the ponderous body-mass, and hence symptoms of heart-weakness appear.

In such, the heart is overtaxed even when the body is in repose, and manifests its debility at all times. In some instances cardiac symptoms first make their appearance after some unwonted exertion or after an attack of influenza or some other acute infectious disease. In a few cases disease of the coronary arteries is responsible for an attack of angina pectoris, or for sudden death, through rupture of the heart-wall. But such conditions are wholly independent of the obesity. This conception of the fatty heart, entertained as it is by two such masters as Leyden and Romberg, is in strict accordance with daily observation, and makes it clear why one enormously fat person is capable of performing a degree of physical effort wholly impossible to another much less obese. It is evident, also, how fallacious it may be to diagnose fatty heart merely on the ground of general corpulence.

Etiology.—The causes of an excessive growth of fat on the heart may be said to be those of obesity in general. There seems to be a marked tendency to corpulence in some families, and their members accumulate fat notwithstanding all efforts to the contrary. Such a predisposition is sometimes witnessed among children; but as a rule corpulence does not manifest itself until after puberty or still later, between the ages of thirty and forty. Age itself is a predisposing factor, particularly with women, who show a striking tendency to increased weight after the menopause.

The female sex in general is said to show a greater inclination to corpulence than does the male sex, yet the difference in this regard is probably to be attributed to differences in occupations and habits, since women generally take less exercise than men. They are, moreover, apt to be chlorotic and anæmic, and it is well known that fat and anæmia often go together, in consequence probably of the diminished oxidizing power of the blood. People of sedentary pursuits are especially liable to take on fat, and with family inheritance and occupation combined, obesity becomes inevitable.

Of all causes, the one most potent next to inherited tendency is consumption of food rich in carbohydrates conjoined with an excessive intake of fluids. Gluttony (luxus consumption) conduces to obesity even though there is not a relative disproportion in carbohydrates. This is especially injurious when added to inadequate exercise. The too free drinking of fluids is another potent factor, and when in the form of malt liquors, fat may be taken on very rapidly. The excessive use of alcohol in any form, moreover, is said to favour the development not only of fat in general, but in particular of the deposit of adipose tissue upon the heart.

The foregoing are the leading causes of fatty overgrowth, but it must be remembered that the modern conception of fatty heart is not necessarily a surplus accumulation of adipose tissue beneath the epicardium and between the bundles of muscle-fibres, but a manifestation of cardiac insufficiency attributable primarily to general obesity. Consequently, in studying the etiology of the heart-weakness exhibited by corpulent people, we must bear in mind what was said above concerning the pathology of the fatty heart. Whatever tends to undermine muscular strength in general produces a weak heart-muscle, and in the obese such influences are specially deleterious.

Luxurious living, indolent habits, excesses of all kinds (including the abuse of tobacco), anæmia, and chlorosis—all tend to produce a flabby heart-muscle. Such a heart is incapable of that driving power necessary to force the blood through the extensive system of capillaries created for the supply of new adipose tissue, in addition to those ramifying in the organs, muscles, bones, etc. Under the demands of a quiet existence such a heart may show no incompetence severe enough to attract the person's attention. When, however, cardiac inadequacy makes its appearance, it is gradual and insidious, or abrupt in consequence of unwonted exertion or of acute illness. In such cases the obesity is the predisposing cause, and the conditions that bring about heart-strain the exciting cause.

Finally, Romberg includes among the causes of cardiac insufficiency a too strenuous anti-fat diet which is practically a starvation diet, and too rigorous depleting measures acting through the skin and bowels. If, in addition, vigorous exercise is taken, the undernourished heart-muscle can readily become overstrained.

Symptoms.—There is nothing in the symptoms peculiar to the disease under consideration. Shortness of breath is usually the first symptom to make its appearance, but such persons are so accustomed to quickening of respiration during exertion that they give no heed to it until it has reached a degree of considerable or continuous dyspnœa. At first, embarrassment of breathing is only noticed during hurry or the effort of ascending stairs, but subsequently it is called forth by the mere act of rising from a chair and walking across the room. Stooping or bending forward is apt to cause great dyspnœa; and as cardiac feebleness progresses, distressing shortness of breath declares itself during the taking of food, and there is panting respiration even during conversation. At length in this, as in other forms of heart-disease, a stage of orthopnœa is reached when dyspnœa becomes habitual, even while the patient is at rest.

Another early symptom in some cases is lightness of the head or vertigo, especially likely to appear when the patient gets on to his feet or changes the recumbent for the upright position. In some instances there are attacks of veritable syncope, the feeble heart failing temporarily to maintain cerebral circulation. It sometimes happens that a patient dies in such a syncopal attack under appearances which caused Stokes to term it "apoplectiform."

Another symptom also observed in the early stage of the disease is acceleration of the pulse. Stokes, Walshe, and other early English writers laid particular stress on slowness of the pulse as a sign of fatty heart, but as a matter of fact it is more common for the pulse to exhibit an increase in frequency. It is also apt to be small and feeble, although associated arterial sclerosis or chronic nephritis may give it undue tension. Another not infrequent feature of the pulse is instability—i. e., a lack of steadiness in its rhythm—fluctuations taking place in its rate without apparent cause. Irregularity in force and volume and intermittence, however, are not common.

In some instances the earliest symptoms are referable chiefly to the digestive organs. The patient finds that his usually small appetite has become still more diminished, or that so soon as he has eaten a little he is oppressed by an uncomfortable sense of fulness and shortness of breath. Unquenchable thirst impels him to drink large amounts of water or tea, which but increase his oppression, and he is annoyed by frequent eructations of gas. His bowels are sluggish and constipated, and his urine becomes scanty and high-coloured. He is apt to fall asleep in the chair, particularly after meals, while at night he is wakeful, or if he sleeps, is harassed by nightmare and dreams. Headaches, usually dull and heavy, but sometimes of a neuralgic character, are not uncommon.

If the circulation has not become too seriously embarrassed, and particularly if the heart-muscle is intact, the symptoms being due to a disparity between the size of the body and the power of the heart, then measures calculated to reduce the obesity and thus restore the proper relation between body weight and heart power may reinstate the patient's health. In very many cases, unfortunately, this is impossible; the heart-muscle has become seriously damaged through atrophy or degeneration or coronary sclerosis, or serious dilatation has been set up in consequence of long years of overstrain, or as the result of some single indiscreet effort. Symptoms of failing circulation now appear and progress steadily, it may be rapidly. Cough and frothy mucous expectoration, attacks of asthma and cardiac pain of an anginoid character, or even of true angina pectoris, are added to the previously existing dyspnœa. Hepatic congestion and tenderness, scanty albuminous urine, and œdema of the ankles are discovered, and before long the patient presents the well-known picture of the final stage of heart-disease which has been so often described in these pages. It scarcely requires physical examination of the chest to convince one that the heart is dilated and overburdened. Orthopnœa compels the patient to remain in his easy chair, and in the hope of obtaining still greater ease, or of lessening the dropsy, the swollen, tense, and shiny legs are supported upon another chair or stool. Nurses stand by his side to administer stimulants, or by fanning him, to mitigate his attacks of dyspnœa. Sleep visits him but fitfully, if at all, and neither day nor night brings him relief from his torment.

In this manner one week merges into another, and he is to be accounted fortunate when pulmonary œdema ends his suffering, or the heart stops suddenly and unexpectedly. It is the same old story over and over, varied only by the greater prominence of some symptoms in one case and of others in another, or by the longer or shorter duration of the struggle.

Physical Signs.—*Inspection.*—Obesity renders examination of the thoracic and abdominal organs difficult and unsatisfactory. If close scrutiny fails to detect cardiac impulse, this must not necessarily be attributed to feebleness of the impulse; it may be due to the intervening layer of adipose tissue.

Palpation.—For the same reason the hand laid upon the chest fails to locate the apex-beat, or indeed to perceive any cardiac shock whatever. The real value of palpation, therefore, is in the study of the pulse, which should be carefully studied for any information it may afford. If it is of good strength and volume and in rate is stable and not unduly accelerated, it points to a fairly healthy heart-muscle. If, on the contrary, the peripheral arteries are thick—a matter which the corpulence often renders by no means easy of determination—if the pulse is unsteady and perhaps intermittent, then it is likely that chronic myocarditis is present or that the muscle-fibres have suffered atrophy from possible encroachment upon them by the excessive deposit of fat. Palpation of the liver with a view to ascertaining if this organ is enlarged, is also a matter of great difficulty and even impossibility, in consequence of the size and resistance of the corpulent abdomen. Even if the liver is palpable, this may be due to its being fatty, and not to a state of passive congestion.

Percussion.—This means of investigation, upon which so much reliance is ordinarily placed for the detection of cardiac enlargement, is of but small aid in the obese, for reasons that lie in their corpulence. There is often a development of fat within the mediastinum which gives an area of dulness that may be thought to belong to the heart, yet in reality does not. Furthermore, the mass of fat within the omentum and upon the abdominal walls impedes the descent of the diaphragm, if it does not actually crowd it upward, and thus cause the heart to assume an abnormally high and horizontal position. When this is the case the area of cardiac dulness is increased transversely and upward, giving a false appearance of increased size of the organ. Consequently extreme care is necessary in drawing any conclusion from an increase of præcordial dulness. If, however, by percussion in the various ways described in the introductory chapter one becomes satisfied that such an increase does not exist, it affords presumptive evidence that the symptoms are due to potential, not structural incompetence.

Auscultation.—This is likely to afford the best evidence of the real state of the heart, and yet we know that the muscle may be seriously diseased without any appreciable change in the heart-sounds. If these are found to be only rather distant and feeble but still clear, and the aortic second sound of good relative strength, it is in favour of the integrity of the heart-muscle being still preserved. If, on the contrary, the first sound is disproportionately feeble, perhaps impure or even obscured by a systolic apex-murmur, if the aortic second sound is weak and the pulmonic second unduly loud, there is reason to believe the heart is enlarged. This may be a simple hypertrophy with dilatation, or there may be in addition myocardial degeneration. Physical examination alone does not enable us to decide; we must endeavour to determine this point by the study of all those factors outside of the heart which make for or against cardio-vascular decay.

Diagnosis.—It is not a difficult matter to diagnose cardiac inadequacy. The real problem to solve is whether the heart is only potentially unequal to its work or is incompetent in consequence of fatty overgrowth or of myocardial disease. If the pulse is normal in rate and quality, and if subjective symptoms are felt only upon exertion, are slight and quickly subside after cessation of effort, the heart-walls are presumably intact. This conclusion is strengthened if minute inquiry fails to elicit history of cardiac strain, acute infectious disease, bad habits, or any other influence that may serve to impair the integrity of the myocardium. On the other hand, degenerative changes are probable if the patient is past middle age, if the pulse shows notable alteration in quality and rhythm, and if symptoms of inadequacy are present even when the patient is at rest or not making unwonted demands on his heart. If the individual belongs to the category of fat and anæmic, the heart-muscle is likely to be flabby and its incompetence to be due to dilatation. If, on the contrary, symptoms of inadequacy develop in the fat and plethoric, whose skeletal muscles are firm and large and whose weight is due to the great specific gravity of their muscles, bones, organs, etc., and not to adipose tissue, it may reasonably be concluded that the heart is overstrained, perhaps dilated, but not hampered by deposit upon it of fat.

Finally, if symptoms of cardiac incompetence develop in any corpulent person it is the part of wisdom to make a diagnosis of cardiac inadequacy and not of fatty heart, for we possess no means of determining during life whether there is or is not an excessive deposit of fat within the heart-muscle.

Prognosis.—This depends upon the condition which is responsible for the embarrassment of circulation. If the patient is young and muscular and the cause of the heart-weakness is found to lie in potential, not structural disability, or if the symptoms date from some recent cardiac strain, the heart-muscle having been previously competent, the prognosis is comparatively favourable, since appropriate treatment may restore compensation. If, on the other hand, the patient's musculature is flabby, he is anæmic, and gives a history of indolent habits; if his symptoms have steadily increased, and especially if their gravity indicates a serious breakdown of the heart—then there is but small hope of reinstating compensation, and death is only a question of time. Angina pectoris, attacks of asthma, thickened arteries with high sustained pulse-tension, likewise furnish a hopeless prognosis as regards recovery. The probable duration of the malady cannot be stated with any accuracy, but the course is likely to be a short one. In other respects, prognosis is governed by the same conditions as in other forms of heart-disease.

Treatment.—It is essential, in the correct management of any disease, that the physician have a clear knowledge of its pathology and of the object to be attained by treatment. If by the term "fatty heart" were meant simply a heart overgrown and infiltrated with adipose tissue, then the plain indication of treatment would be the absorption of excessive fat, and the object would be accomplished by putting the patient upon a *régime* calculated to reduce his obesity. In this chapter, however, the disease has been considered from a different standpoint. It has been looked upon as a condition of potential weakness, the heart becoming relatively inadequate to the requirements of the circulation, rendered necessary by the size of the body. There may or may not be an undue deposit of fat upon the heart itself. This being the pathology of the disease, the indication is to restore or establish a proper relation between cardiac power and body weight. This is to be accomplished by measures that will either invigorate the heart-muscle without reduction of the obesity, or will bring about the latter without the former, or will do both. By the patient, it is generally thought that the reduction of his corpulence is all that is necessary; but Romberg repeatedly utters an emphatic warning against such an idea. He states again and again that harm rather than benefit is likely to follow the indiscriminate employment of the ordinary anti-fat cures, since they increase the already existing heart-weakness. The need of such a warning was forcibly impressed upon me only this past winter.

A corpulent man of fifty-five, who had yet been able to exercise without special discomfort, concluded he would try a reduction-cure at Marienbad, Germany. By vigorous use of the waters and an unreasonable amount of walking he reduced his weight 45 pounds in a few weeks and returned home feeling, as he said, " fine." Nevertheless, he had not been home long when, on attempting to walk to his place of business one morning as usual, he was seized with great shortness of breath, that compelled him to return to the house. This was the beginning of the end, for he failed steadily in spite of the most approved treatment, and died in less than six months.

Depleting measures should be confined to cases in which circulatory disturbance is attributable to obesity and not to cardiac insufficiency. Such cases are found for the most part in persons who are still young, or have not yet passed the age of forty. It is often a matter of great difficulty to determine whether the trouble resides in the heart or not, and therefore an anti-obesity plan of treatment should not be decided on hastily or without thoughtful study of all those considerations bearing on this point. Should this plan of management be at length decided upon, the effect on the heart should be carefully watched and the treatment discontinued altogether, or the weight reduction carried on less vigorously, so soon as debility, nervousness, and other signs of cardiac or general asthenia make their appearance.

It is of importance also what system of dietary is selected. There are several well-known anti-fat dietaries, such as Banting's, Ebstein's, Oertel's, and Sweninger's, but they all have the one feature in common, that they greatly restrict the consumption of carbohydrates. Their chief differences are in the amount of albumin and fat allowed. Ebstein permits much less albumin and far more fat than do the others, while the Oertel system allows considerably more albumin and far less fat, and again more carbohydrates. They all restrict the consumption of fluids. Whatever differences they possess, they all attain their end by causing an absorption of fat, both by the taking away of fat-forming food and, with the exception of Ebstein's, by the administration of a relatively large proportion of albumin, which is thus said to stimulate the absorption of fat. In addition to restricting the diet, exercise is insisted upon and saline cathartic waters are administered.

The great objection to the employment of such a *régime* in cases of so-called fatty heart lies in the fact that unless the individual is capable of considerable exercise, whereby adipose tissue may be oxidized, the obesity will only yield when the diet is so strict as to become practically a starvation diet. It is a well-known principle underlying the dietary of heart-disease, that inasmuch as the heart-muscle performs an enormous amount of work, it should receive a relatively large proportion of proteid—i. e., tissue-forming food—and must under no circumstances be deprived of adequate nourishment. Consequently, if an attempt is to be made to diminish the corpulence of a person with cardiac insufficiency, a dietary must be selected that will most nearly meet the demands of the heart. This is undoubtedly the one selected by Oertel, while the Ebstein and Banting systems are clearly inadmissible. The daily allowance of the several elements permitted by Oertel are as follows: Albumin, 5½ ounces; fats, 1 to 1¼ ounces; carbohydrates, 2½ to 3½ ounces. For particulars the reader is referred to Oertel's original work, or to treatises on dietetics.

In carrying out a diet for the reduction of obesity in the class of cases now under consideration, it is very unsafe to produce a too rapid loss of weight. In my opinion this should not exceed 2, or at the very outside 3 pounds a week, and in many cases 1 pound is better. Consequently the physician should keep an accurate record of the weight, and many times will have to modify the diet given above by increasing the albumin or starches, or both. If the physician is in doubt concerning the actual state of the heart-muscle, or if the patient finds he is unable to take adequate exercise, then massage will often be found of great service by promoting oxidation of adipose tissue. It also aids the circulation.

The daily use of laxative waters is essential, and Germain Sée recommends the administration in moderate doses (5 to 10 grains) of iodide of potassium three times a day.

For the past few years the public and profession have heard much concerning the efficacy of reducing fat, of the alternate daily use of Vichy and Kissingen waters. From my rather limited observation of their effects, I am inclined to the opinion that if these waters are to prove efficient they must be combined with exercise and at least a moderate restriction in the consumption of carbohydrates.

In elderly people, or those with feeble muscular development, or in such as already display pronounced symptoms of cardiac inadequacy, energetic treatment for the reduction of corpulence is hazardous, to say the least. In many instances the weakness of the heart will be intensified. Romberg is of the opinion that such persons should not be subjected to the possible dangers of such treatment; while to make a routine practice of depleting all fat patients certainly cannot be too strongly condemned. Nevertheless, I believe in most cases, even when the heart is primarily at fault, some modification of the diet will usually prove beneficial. Some of these patients are anæmic as well as corpulent—some because they have been light feeders for years, others because they have habitually taken too little albumin and too much starch and sugar, while still others have consumed altogether too much fluid, particularly at meal-time.

In the first class, attempt should be made to secure more adequate nourishment through medicinal or other measures calculated to improve the appetite and assimilation. To this end simple bitters and tonics—as quassia, gentian, iron, nux vomica, arsenic, or the hypophosphites—may be tried, together with acids, pepsin, pancreatin, and kindred preparations. A cupful of hot water half an hour before each meal often improves both appetite and digestion. In quality the meals should be highly nutritious, so that in quantity they may be light. This may be accomplished by the addition of the expressed juice of fresh beef, or by some one of the prepared foods rich in nitrogen and fat but poor in carbohydrates.

For the second class it can do but little harm to reduce starches and sugar and increase the animal food, without, however, conforming strictly to the amount and proportion laid down in rigid anti-fat dietaries. In the last class it may be sufficient to diminish the ingestion of fluids without otherwise curtailing or modifying the food allowance.

In all individuals who display more or less heart-weakness the important point in the management must be the attempt to re-invigorate the heart. If its load cannot be lightened—that is, if the corpulence cannot be reduced—efforts to strengthen the heart are likely to prove futile. The physican will then have to choose one or the other alternative; either to persevere in his futile attempt to rehabilitate the heart, or to run the risk of reducing the body weight. The wise thing will be to try to accomplish both. It may be that the loss of half a pound or a pound a week will not materially weaken the patient, and yet may be sufficient to greatly aid the doctor's efforts towards re-establishing cardiac power.

By all odds, the best means to this end is exercise. This should be limited to two kinds—easy walking and resistance gymnastics. Rules for the latter have already been given (see page 455). The conditions that are to control the daily walk should be carefully laid down by the physician. (1) Walking should be done from one to four hours after meals, according to the degree of cardiac debility. (2) The walk should not be so prolonged as to occasion fatigue, and of course must vary greatly in individual cases. The medical adviser will have to determine its length by observing the immediate effect of exercise, or by a searching inquiry as to symptoms. (3) The pace must not be fast enough to cause shortness of breath or palpitation, and it is always best to begin very slowly, the gait to be quickened only as the exercise produces a feeling of well-being or lightness in the chest. (4) The patient must not walk against a strong wind, and must confine his exercise to level ground. Attempts to carry out the Oertel system of ascending an incline are not to be permitted until a considerable degree of compensation has been established. If the patient is exceptionally intelligent and his judgment can be relied upon, it may be safe to allow him a little latitude in this regard; but patients are more likely to do themselves harm by essaying paths that are too steep, than they are to derive benefit from accustoming themselves to ascending gentle acclivities. Therefore in a large majority of instances the fourth rule should be strictly insisted upon.

When heart-weakness has reached such a degree that walking even about the room occasions decided dyspnœa, there is no prospect of improvement from exercise, and life will probably be prolonged by keeping the patient quiet and relying on skilful massage or very carefully conducted gymnastics. Another highly useful and often very promising measure for restoration of heart-power is the balneological treatment—i. e., saline baths as given at Bad Nauheim and already described. In the hands of one experienced in their use these baths are rarely likely to do harm, except in those cases in which dilatation has become extreme, or other contra-indications are present.

Among therapeutic measures are included also the ordinary heart-tonics, such as digitalis, strophanthus, and their congeners, as well as strychnine and cardiac stimulants, nitroglycerin, ammonia, camphor, and valerian. The same rules govern their administration in these as in other cases of cardiac insufficiency from whatever cause. Romberg is of the opinion that but little good is to be expected from digitalis; but in my opinion, if its vaso-constrictor effects can be counteracted by nitroglycerin or iodide of potassium, the remedy should theoretically support the failing heart in cases of obesity, as well as in any other nonvalvular disease. If pulse-tension is persistently high, strophanthus may be of use, either alone or combined with digitalis and strychnine. The last-named heart-tonic should never be omitted.

In all cases of obesity blood-pressure is high within the abdominal vessels, and therefore I firmly believe that if any results are to be attained from the use of heart-tonics, or indeed from other measures, as exercise and baths, tension within the abdomen must be lessened by the persistent use of cathartic remedies. Both because of the tendency of alkalies to reduce weight, and on account of their non-irritating properties, the cathartic selected should be a saline aperient water—Hunyadi, Rubinat-Condal, Concentrated Pluto, Apenta, Franz Josef, Carlsbad, or any other of the well-known aperient waters on the market. Care should be had not to produce weakness by a strongly purgative effect each day, but only to keep the stools semi-liquid and copious.

It is usually well to introduce a dose of calomel or blue pill occasionally at bedtime. The compound infusion of senna, which is only the old English "Black Draught," 4 ounces of which may be taken at a time, forms a capital purgative for occasional use.

Special management is required by complications, as, for example, the use of iodide of potash or soda in chronic arteriosclerosis, sometimes observed in obese patients, nitroglycerin and morphine in cases of angina pectoris or cardiac asthma. When at last cardiac power is utterly lost, diuretin-Knoll may be of service in reducing dropsy, or at least holding it in check. Overdistention of the cardiac cavities, particularly the right chamber, may be temporarily relieved and the patient's suffering ameliorated by venesection. Owing to the associated anæmia, the amount of blood thus abstracted should be small, 6 to 12 ounces being usually sufficient to render the pulse soft and full. Other measures for the relief of the patient must be left to the physician's judgment and to the special indication of each case.

CHAPTER XXIV

CARDIAC ASTHMA—CHEYNE-STOKES RESPIRATION—BRADYCARDIA—STOKES-ADAMS DISEASE

I. CARDIAC ASTHMA

No one symptom is so frequent a feature of cardiac disease as dyspnœa, and with the possible exception of præcordial pain there is no subjective disturbance so distressing to the patient. In many instances, moreover, it occasions such obvious distress as to be actually harrowing to the spectator. For the most part, shortness of breath is the result, and hence an expression, of mechanical impediment to the circulation. Conequently it forms an important part of the clinical picture in all forms of cardiac inadequacy.

It would be a mistake, however, to attribute the dyspnœa of cardiopaths solely to circulatory embarrassment. Thus it may be due to pain, in consequence of which the individual fears to breathe with his customary depth and slowness. In other cases it may result from nervousness or apprehension, as e. g., during an examination of the heart. In all such instances, however, it is usually easy, by giving due consideration to the state of the circulatory apparatus, to recognise the true cause of the breathlessness.

Cardiac dyspnœa is *par excellence* a dyspnœa of effort—i. e., it is either evoked by exertion or intensified by the same. This breathlessness of effort may be regarded as the earliest manifestation of failing heart-power, and so long as cardiac incompetence is of minor degree, is confined to periods of physical exertion. There nearly always comes a time, however, when dyspnœa becomes more or less constant even during rest and when apparently trivial conditions intensify the shortness of breath even to the point of positive air-hunger. This has been repeatedly dwelt upon in foregoing pages in considering the manifestations of valvular disease.

Persons suffering from myocardial inadequacy of whatever cause also display dyspnœa of effort quite like that of other cardiopaths, and likewise due to circulatory embarrassment.

There is a form of dyspnœa displayed by these patients, however, which is so intense and paroxysmal that it has not inaptly been termed cardiac asthma. As implied by the name, it closely resembles an attack of bronchial asthma. In most cases it is not a growing intensification of already existing dyspnœa, but is a more or less sudden attack of such distressing shortness of breath as to constitute veritable orthopnœa for the time.

The attack may be induced by effort, but in its most typical form it comes on at night. It is therefore a nocturnal dyspnœa. The attack may seize the individual so soon as he lies down at night, but frequently it does not appear until after he has been asleep for a few hours. The patient is then aroused by a sense of oppression or want of sufficient air, which obliges him to sit up or arise and walk slowly about his apartment.

In its mildest manifestations this is all, but generally the dyspnœa is far more severe. The shortness of breath increases until in a few minutes, occasionally from the very start, the sufferer is forced to breathe with great rapidity and difficulty. His chest emits a multitude of fine or coarse moist râles due to intense pulmonary congestion and transudation of serum into the air-tubes, and the consequent cough is attended with the expectoration of frothy or even bloody mucus.

The patient's distress is now terrible both to himself and friends, his face becomes cyanosed and bedewed with perspiration, while his pulse is rapid, extremely feeble, and even irregular or intermittent. If the heart is now examined it is found to be dilated, while its sounds are extremely faint, partly in consequence of the râles of pulmonary œdema, but mainly because of cardiac weakness.

Such an attack may last for minutes or even hours, yet with scarcely the initial severity. As a rule it abates in from fifteen to thirty minutes. With cessation of the terrific dyspnœa the sufferer is left exhausted and usually in a state of great mental agitation.

The cause of this cardiac asthma is believed to be temporary weakness of the left ventricle and disproportionate strength of the right ventricle. This condition on the part of the two ventricles leads to congestion of the lungs and consequent dyspnœa. As the stasis increases and pulmonary œdema occurs, dyspnœa becomes increased in consequence of mechanical interference with oxygenation of the blood. Certainly such an explanation fits the clinical manifestations of an attack.

The predisposition to cardiac asthma is furnished by degeneration and enfeeblement of the left ventricle, while the immediate or exciting cause may be found in whatever temporarily overpowers the ventricle—i. e., undue physical effort. Coitus, by reason of the union of both effort and excitement, seems particularly apt to excite an attack. The occurrence of the attack after some hours of sleep is thought to be explained by the augmentation of blood-pressure said to be incident to the recumbent posture. Huchard states that blood pressure is increased by the recumbent position, while Gaertner, on the other hand, claims that his tonometer shows an actual though slight decrease of pressure. If this is so, some other explanation is required for the occurrence of cardiac asthma during sleep. This may be found in the added work put upon the left ventricle in maintaining blood-flow by muscular inaction, and the more quiet respiration incident to sleep.

It is needless to remark that such attacks are highly dangerous and call for prompt and energetic treatment. To this end stimulants are indicated, and nothing is so efficient as the hypodermic injection of $\frac{1}{8}$ of a grain of morphine combined with the atropine found in the ordinary hypodermic tablet.

II. CHEYNE-STOKES RESPIRATION

This is a rhythmical form of dyspnœa, first carefully described by the two eminent physicians whose names are now inseparably linked with this distressing symptom. It is characterized by alternating periods of dyspnœa and apnœa, which recur at regular intervals and supplant normal breathing.

The phenomena of this type of respiration may be described as follows: After a period of suspended breathing or apnœa, respirations return, at first slowly and superficially, each succeeding one quicker and deeper, until at length the inspirations become strikingly rapid and forcible, and maximum dyspnœa is attained. After having maintained this height for a few seconds the respiratory efforts begin to abate both in speed and depth, dying away gradually into another period of complete repose, the chest being now entirely motionless. After this stage of apnœa has lasted for a certain number of seconds, breathing again begins in the same scarcely perceptible manner to wax and wane as before. Thus cycle after cycle is repeated with perfect regularity.

The duration of the two phases that constitute an entire cycle or paroxysm differs in different cases, but remains constant in the same case after this form of dyspnœa has become well established. It is not very uncommon to witness incomplete or abortive manifestations of this peculiar dyspnœa, which after a time assume the typical Cheyne-Stokes respiration. Apnœa usually persists for a quarter to half a minute and the period of dyspnœa a few seconds longer. In 12 cases observed by Sansom the pause varied in duration from ten to forty seconds and dyspnœa from fifteen to fifty-five seconds. In one case he noted a persistence of apnœa for sixty and of dyspnœa for sixty-five seconds, while he cites another in which each phase continued two minutes. During the stage of respiratory effort the ascending and descending series endure about twenty seconds each, while the acme of laboured breathing continues about twenty-five seconds (Sansom). The length of this period—i. e., of dyspnœa—is not the same in all cases, however.

Patients exhibiting Cheyne-Stokes respiration usually retain consciousness during their attacks and suffer no enfeeblement of their mental powers. In some, on the contrary, consciousness is clouded and the intellect impaired.

Certain pupillary phenomena have been observed. Thus the pupils are contracted during apnœa and dilated in the stage of dyspnœa. Finlayson has described a rhythmical contraction and dilatation of the pupil " during the acme of the respiratory act," dilating with inspiration and contracting with expiration.

Such rhythmical change in the size of the pupils is not confined to this form of dyspnœa, however, for a similar phenomenon has been observed during the stertorous breathing of coma.

As regards the pulse, very conflicting observations have been recorded. Some observers have reported perfect uniformity in the pulse-rate during both periods of the cycle, while Little, quoted by Sansom, witnessed 15 heart-beats during an apnœal period of ten seconds, and only 6 in a like period in the dyspnœal stage. Others have reported retardation of the pulse during apnœa, and a relative acceleration during the period of dyspnœa. Aside from changes in rhythm, the tension of the pulse is said to be raised, the pulse feeling harder and firmer than normal.

Diseases in which Cheyne-Stokes Breathing is Observed.—The following list is taken from Sansom's work on the Diagnosis of the Diseases of the Heart and Thoracic Aorta, and shows that the cases in which this form of dyspnœa is observed are by no means exclusively those of cardiac disease. (1) Cases attended with cerebral affections—viz., cerebral hæmorrhage, tumours, tubercular meningitis, epilepsy, shock from surgical injury with uræmia, alcohol intoxication, opium poisoning, and insanity. (2) Cases attended with lesions of the heart and great vessels—viz., fatty degeneration of the heart, pericarditis, atheromatous disease of the aorta, aortic aneurysm, valvular disease (double aortic, with mitral insufficiency, mitral stenosis, dilatation of aorta coexisting, aortic regurgitation and obstruction), and chronic Bright's disease. It is with diseases of the aorta and its valves that it is most frequently associated, but it may occur, in the absence of valvular disease, when the coronary arteries are obstructed. In any of these conditions it is most probable that the arteries at the base of the brain are atheromatous, and the concurring affections of the heart and brain speedily lead to death. (3) Cases of certain acute febrile diseases—viz., diphtheria (Hütterbrenner), typhoid fever (Wharry), puerperal septicæmia, scarlet fever, pneumonia, pertussis (with inanition), and influenza.

Theories to Explain Cheyne-Stokes Respiration.—Before giving a brief statement of the leading theories which have been advanced to explain the rhythmical alteration of breathing, it may be well to state certain physiological facts concerning respiration. (1) Inspiration is a result of the contraction of the inspiratory muscles in response to a nervous impulse sent out from the respiratory centre in the upper portion of the medulla oblongata, close to the calamus scriptorius, but extending to the upper portion of the spinal cord. (2) Expiration is for the most part a passive act due to the elastic resilience of the lungs. (3) The action of the respiratory centre is automatic and rhythmical. (4) The activity and energy of the respiratory centre depend in great measure upon the amount of oxygen contained in the blood, and upon the amount of blood supplied to the centre. It is not difficult to understand why there may be dyspnœa in any given case, but it is difficult to explain why the dyspnœa should be rhythmical in the way characteristic of Cheyne-Stokes breathing.

The first attempt to explain it was made by Traube, and is known as *Traube's theory.* This assumes that the normal excitability of the respiratory centre is diminished in consequence of the supply to it of imperfectly oxygenated blood. During the stage of apnœa carbonic acid accumulates in the blood, and when it has become excessive begins to stimulate the respiratory centre to discharge its impulses. In response to these discharges, which are at first slow and imperfect, contraction of inspiratory muscles takes place, grows ever deeper and more rapid until at length the maximum stage of dyspnœa is attained. The centre now ceases to be stimulated, or becomes exhausted, and inspiratory efforts gradually decline until they finally terminate in the stage of respiratory pause or apnœa. Carbonic acid in the blood is again accumulated, the respiratory centre is again stimulated, and thus the cycle is repeated in ever-recurring paroxysms.

Many objections have been urged against Traube's theory, but the one that Bramwell thinks is fatal to it is that a deficient supply of properly oxygenated blood to the respiratory centre would stimulate it into action rather than impair its irritability, since it is not so much an accumulation of carbonic acid as a want of oxygen in the blood which stimulates the respiratory centre.

To explain the lowered irritability of the respiratory centre, which is assumed in Traube's theory, Sansom has advanced the proposition that the centre is in a state of paresis or partial paralysis in consequence of some cerebral disease, and with a satisfactory explanation of the diminished excitability of the respiratory centre Traube's theory would then become complete.

Filehne's Theory.—This assumes that both the vaso-motor and respiratory centres are concerned in the production of this form of dyspnœa. According to his explanation, the deficiency of oxygen and excess of carbonic acid in the blood, which result from the period of apnœa, stimulate the vaso-motor centre, and the arterioles of the brain, as well as those throughout the body, become contracted. This constriction of the arterioles diminishes the supply of blood to the respiratory centre, and in consequence this centre is stimulated to discharge, and inspiration begins. So soon, however, as respiration has become energetic and the blood properly aerated, stimulation of the vaso-motor centre ceases, arterial spasm is no longer maintained, the respiratory centre receives a proper supply of arterialized blood, and dyspnœa is no longer experienced. The respiratory acts gradually die away and the period of apnœa is again reached. There again occurs stimulation of the vaso-motor centre, and another cycle is repeated. Bramwell is of the opinion that if Filehne's theory is correct, then Cheyne-Stokes breathing should occur much more frequently than it really does. He says: " I am disposed, therefore, to think with Dr. Sansom that something more is necessary, and that there must be some alteration of the respiratory centre itself in addition to the condition which Filehne's theory supplies. A state of irritable weakness would, in my opinion, account for this condition."

Bramwell's theory in explanation of Cheyne-Stokes breathing is based on the supposition that the respiratory centre consists of two parts: an inspiratory and an expiratory, and that, as suggested by Rosenthal, " the inspiratory centre is the seat of two conflicting forces, one tending to generate inspiratory impulses, (the discharging portion of the inspiratory centre as we may call it), and the other offering resistance to the generation of these impulses (the restraining or inhibiting portion of the inspiratory centre)—the one and the other alternately gaining the victory, and thus leading to rhythmical discharge." Bramwell assumes that venous blood excites the discharging portion, restrains the inhibiting portion; while oxygenated blood depresses the former portion, and intensifies the action of the restraining portion. If the discharging portion is in a condition of irritable weakness, and therefore more easily excited to discharge, but also more quickly and easily exhausted, or if both portions are in a condition of irritable weakness, then there is a condition of things, Bramwell thinks, which satisfactorily explains the phenomena of Cheyne-Stokes breathing. At the end of apnœa the blood is highly venous, and therefore gradually excites a paroxysm of dyspnœa, by stimulating the discharging and restraining the inhibitory portion of the centre. In the second place, the carbonic acid in the blood stimulates to action the vaso-motor centre, the arterioles become contracted, and the supply of oxygen to the respiratory centre is still further diminished. Furthermore, the irritable weakness of the discharging centre causes its impulses to become excessive, and the state of dyspnœa results. Moreover, the weakness of the discharging portion of the inspiratory centre causes it to become quickly overexhausted and the dyspnœa subsides. In consequence of the energetic respiratory effort during the stage of dyspnœa the blood becomes arterialized and the discharging portion of the inspiratory centre is no longer stimulated, but the reverse takes place as regards the restraining portion, which gains the ascendency over the weakened and exhausted discharging portion, and the state of apnœa is produced. During this period of rest the oxygenated blood, which had stimulated the restraining and depressed the discharging portion of the inspiratory centre becomes replaced by carbonic dioxide; the discharging centre is aroused into action again, and the inhibiting is restrained; inspiratory efforts are renewed and another cycle is repeated.

Of the foregoing theories, conceived to account for this distressing rhythmic form of dyspnœa, Bramwell's is the most satisfactory, and yet, as he himself suggests, it is difficult to explain how this condition of irritable weakness of the respiratory and vasomotor centres is produced. Bramwell assumes that in those cases of Cheyne-Stokes breathing displaying a contracted pulse and pallid countenance, there is local anæmia of the centres in consequence of arterial spasm, and irritable weakness takes place.

In other cases not showing arterial spasm he suggests that this unstable state of the centres may be due to disease within the medulla or to impressions received from nervous centres situated higher up or from the periphery, especially from the heart or lungs, through the agency of the pneumogastric and superior laryngeal nerves. Such peripheral stimuli are particularly likely to be received by the centres in those cases of heart-disease manifesting right-ventricle dilatation with diminished supply of blood to the lungs.

Rosenbach's Theory.—After, as he states, a searching analysis of the various theories, Rosenbach has adopted the following explanation. Under the influence of certain anomalies of brain-nutrition there develop localized disturbances in the brain or in individual centres, particularly in that of respiration, which disturbances lessen the excitability of the affected part and augment the normal exhaustibility of the same. Thereby are produced remissions in the activity of the respective centres with loss of tone in the vaso-motor and vagus centres, or complete intermissions, such as a pause in the respiratory act, with a kind of paralytic state of the cerebrum, manifested by a periodic sleep with contraction of the pupils and movements of the eyeballs. So soon as the fatigue and exhaustion of the centre have disappeared in consequence of cessation of respiration and an augmented internal activity, and its excitability returns, respirations again set in and continue to increase, because the excitability of the nervous apparatus grows out of proportion or waxes more rapidly than the stimulus to activity wanes in consequence of organic work. So soon now as the abnormal exhaustibility of the centre again begins to be felt, it supersedes the stimulus, and therefore the functional activity of the centre lessens, and finally ceases altogether when at last the centre has become completely exhausted. Whether or not respiration takes place is determined by the ability of the centre to respond to stimulus, and the depth of the respiratory act depends not upon the strength of the impulse, but on the functional capability of the nervous apparatus. He thinks that of the various nervous centres the respiratory is the one that suffers most readily and often alone, while the vaso-motor centre is relatively much less frequently affected, and paralysis of this means death.

He furthermore thinks that a regularly intermitting pulse, pulsus bigeminus and alternans, may be a manifestation of periodicity in the function of the vagus and vaso-motor centres in certain cases of nutritional disturbance of the brain, and are analogous to the Cheyne-Stokes phenomenon. As Rosenbach states, this explanation of this abnormal type of breathing differs from others in the assumption, not of a periodic alteration of the stimulus, but in a rhythmic change in the excitability of the centre which presides over respiration, even to a complete abeyance of its function for the time being. He assumes that this rhythmical periodicity as regards excitability is to be referred to some peculiar characteristic inherent in the nervous apparatus by virtue of which it is capable of being exhausted and again aroused to activity.

It is needless to add that, however ingeniously the pathology of Cheyne-Stokes respiration may be speculated upon, the subject is still enveloped in great obscurity.

Prognosis.—The development of Cheyne-Stokes breathing is generally held to be of unfavourable significance, by indicating that a fatal termination is not far off. Yet weeks or even months may sometimes intervene between the appearance of this symptom and death. Murri reported a case in which the phenomenon persisted for forty days, and Sansom one for one hundred and eight days. In the Lancet of April 5, 1890, is the report of a case of a man of ninety-two who manifested the symptom for several years. This type of dyspnœa has also been known to appear, then cease, and reappear after a lapse of several months. In most of the cases that recover, or in which the symptom is greatly protracted, the disease upon which it depends is either some brain-lesion or an acute affection, as influenza. When Cheyne-Stokes breathing is observed in cardiac patients, the underlying malady is itself of a grave nature, and the occurrence of this symptom usually portends a not distant termination of the case. To this rule there are exceptions, however. In April, 1895, I was consulted by an old gentleman of eighty who manifested this symptom. He had pronounced thickening of the peripheral arteries, a greatly hypertrophied and dilated heart, a harsh bruit along the course of the aorta, and a remarkably intense and metallic aortic second sound. In addition to his arteriosclerosis and myocardial degeneration, his liver was cirrhotic and the urine gave evidence of chronic interstitial nephritis. Cheyne-Stokes dyspnœa was typical, and in consequence a well-known Chicago consultant had given a sombre prognosis on the ground that he had never known this symptom to endure for more than three weeks in such cases. Yet *pari passu* with improvement in cardiac tones the dyspnœa gradually abated, and after about two weeks was entirely lost, never again to return during the two years that this patient was spared to his family.

Another gentleman of seventy-one displayed this form of breathing, rather irregularly by day but typically by night, during the time, in which cardiac asthenia was marked, yet recovered from it with gradual improvement in his condition.

It has seemed to me that when Cheyne-Stokes respiration is more pronounced, or perchance is manifested only during sleep, it is not of so grave a prognosis as when present with equal intensity both waking and sleeping. Murri, and recently Pembrie, have called attention to a physiological Cheyne-Stokes respiration observed in healthy persons during sleep. But the patient of seventy-one was not healthy, and therefore the nocturnal manifestation of Cheyne-Stokes dyspnœa during his periods of unconsciousness in sleep could not be regarded as physiological. Finally, the prognosis must be looked upon as specially grave in those cases which also manifest obscuration of the mental faculties.

Treatment.—When Cheyne-Stokes dyspnœa is a symptom of cardiac disease the treatment must be essentially that of the underlying condition. Yet we are called on to mitigate the patient's distress so far as this is possible. This is best accomplished by the hypodermic administration of morphine, which, if it does not remove the dyspnœa, blunts the patient's sensibility. The value of morphine in this class of cases has been the subject of some contention in Germany. At the meeting of the Congress for Internal Medicine at Wiesbaden in 1892, Unverricht read a paper in which he expressed the decided opinion that morphine and atropine are powerless for the removal of Cheyne-Stokes breathing. Other observers have gone so far as to assert that morphine intensifies rather than relieves this symptom. Stadelmann made 25 observations upon the effect of morphine and atropine, alone and combined, upon this type of breathing. The observations were made upon two patients, and the doses were 0.01 to 0.02 ($\frac{1}{6}$ to $\frac{1}{3}$) of a grain of morphine, and 0.001 to 0.0015 ($\frac{1}{60}$ to $\frac{1}{40}$) of a grain of atropine. The effects were neither uniform nor constant. They sometimes shortened the period of apnœa, sometimes that of dyspnœa, and at other times they lengthened one or the other or both. In 5 experiments morphine lessened or removed the Cheyne-Stokes respiration, and in 4 it aggravated the symptom.

Although Stadelmann's observations were so inconstant and unreliable as to the effect of morphine that they seemed to confirm Unverricht's assertion, he nevertheless concluded that on the whole the effect of this agent was to mitigate the severity of the attack. Morphine certainly seems to exert no injurious effects; and since it undoubtedly blunts the patient's sensibility and induces sleep, there can be no contra-indication to its employment, even if it seems occasionally to change an irregular or unperiodic form of this dyspnœa into the periodic rhythm characteristic of Cheyne-Stokes respiration.

A word of caution should be spoken, however, regarding its use in these cases. This symptom is usually observed in elderly individuals with stiffened arteries and degenerated hearts, and as the kidneys very commonly participate in this pathological process one should bear in mind the possibility of these patients being more profoundly affected by the morphine than is desirable or even safe. For this, as well as other reasons, one should employ the smallest dose that will render the patient comfortable. In my experience this is generally $\frac{1}{8}$ of a grain, an amount which I have rarely found necessary to exceed. In this dose the remedy is also a powerful cardiac stimulant, and as such beneficial to this class of patients.

III. BRADYCARDIA

Bradycardia and brachycardia are terms applied to an abnormally slow pulse-rate—that is, to one of less than 60 beats to the minute. Allbutt in his system of medicine objects strenuously to their employment on the ground that, as slowness of the pulse is but a symptom, they are likely to mislead the student by seeming to raise the symptom to the importance of an independent disease. Nevertheless the term bradycardia has come to be so generally used that I have thought best to follow the custom of most writers and give it special consideration. I know by experience that practitioners not only regard it with apprehension, but are often at a loss to account for it, and consequently seek for a statement of those conditions in which it occurs and for an explanation of its significance.

Slowness of the pulse may be either physiological or pathological. A normal pulse-rate of less than 60 is occasionally observed, but when it is as slow as 30 or 28, of which instances have been reported, it becomes a truly remarkable phenomenon. Napoleon Bonaparte is often cited as an instance of a physiologically slow pulse, having had only 40 heart-beats to the minute. It has been thought by some that he was a victim of epilepsy, and that his bradycardia was explicable on that ground. Physiological bradycardia is very exceptional, yet when encountered is not to be regarded as anywise likely to affect the general health.

Osler states that slowness of the pulse sufficient to merit the appellation of bradycardia is sometimes a family peculiarity. Under physiological bradycardia must also be included those instances sometimes yet very rarely observed in connection with hunger and cases of transient slowing of the pulse said by Blot to be seen in about 25 per cent of women during the puerperium. In such cases the rate may sink to 44 or even to 34. Allbutt has noted bradycardia in a healthy man of forty-nine given to excessive sexual indulgence, and has likewise seen it in children as a result, he thinks, of masturbation. In his own case his pulse-rate fell to 48 and to 44 in consequence of exhaustion, for it was restored to its normal rate after a refreshing sleep.

Romberg, in writing on diseases of the heart in Ebstein's Practice, displays characteristic German exactitude by limiting his consideration of bradycardia to cases associated with cardiac disease. This appears to me to be too exact, since slowness of the pulse may by the ignorant be thought to indicate heart-disease. I have decided, therefore, to enumerate the diseased conditions of whatever kind which, according to Riegel, may be associated with abnormal retardation of the pulse. As a basis for his classification he made a study of 1,047 cases in which a pulse-rate of less than 60 was observed. (1) Bradycardia may occur during convalescence from acute infectious diseases, as pneumonia, diphtheria, typhoid fever, erysipelas, and acute rheumatism. Sansom also includes influenza among the acute disorders capable of producing slowness of the pulse, an observation in which Allbutt concurs. It is believed that exhaustion is the cause in such cases. (2) Riegel observed this symptom in 379 cases of disorders of the digestive organs, as chronic dyspepsia, gastric ulcer, cancer, and icterus. The occurrence of a slow pulse in cholæmia is a matter of frequent observation. Grob is also said to have seen bradycardia in connection with œsophageal cancer and typhlitis. (3) The phenomenon under consideration is sometimes met with in diseases of the respiratory organs, particularly emphysema and (4) in diseases of the heart and blood-vessels, specially degenerations of the myocardium depending on coronary sclerosis, atheroma of the aorta (Sansom), but is not frequent in valvular defects unassociated with other alterations of rhythm. In 1 recorded case embolism of a coronary artery was attended with a pulse-rate of 8 to the minute. (5) Bradycardia is occasionally seen in acute nephritis, in uræmia, and was seen in 1 case of hæmaturia (Sansom). (6) Aside from uræmia, bradycardia may be produced by other poisons, as lead, tobacco and coffee, alcohol and digitalis. (7) It is sometimes seen in cases of diabetes, chlorosis, and anæmia. (8) Apoplexy, epilepsy, brain tumours, diseases of the medulla and of the cervical portion of the spinal cord, paresis, melancholia, mania, are all said to sometimes be accompanied by slowness of the pulse. (9) It is sometimes seen in skin disease, affections of the genitalia, insolation and exhaustion from whatever cause.

Finally, with regard to the pathology of bradycardia it may be of interest to give the following summary of Regnard's conclusions presented in a doctoral thesis in July, 1890, entitled Étude sur la pathologie du pouls lent permanent. He is of the opinion that every chronic lesion which causes irritation of any portion of the moderator apparatus of the heart may suffice to produce permanent slowness of the pulse and give rise to the aggregate of symptoms. Such nervous irritation may have many causes, as local anæmia through the influence of atheroma on the peripheral circulation and blood-supply to the nerve-centres, deficient blood-supply to the bulbous portion of the pneumogastric, tumours of the meninges of the bulb, or in the mediastinum acting on the vagus, morbid excitation of the laryngeal and gastric branches of this nerve, but most frequently some affection of the heart itself, as fatty degeneration or coronary sclerosis.

The predisposing conditions are stated to be arteriosclerosis, whether syphilitic, alcoholic, gouty, or rheumatic in origin.

It is not within the scope of this work to consider the significance of bradycardia in other conditions than of the circulatory apparatus. In these conditions marked slowing of the pulse is generally regarded as of serious import, because it is most commonly observed in cases of sclerosis of the aorta or coronary arteries, and in such the heart-walls are likely to be degenerated. The lengthening of diastole incident to slow cardiac contractions subjects the heart to the possibility of diastolic arrest and the patient to the possibility, therefore, of sudden death in syncope. Moreover, the heart-muscle is extremely feeble in such cases, and hence it may require very little additional strain or depression to bring it to a standstill.

I have notes of an old man with rigid arteries and chronic myocarditis in whom for several years prior to death the pulse-rate was persistently about 28. In another the heart was actually slow, but as only every other systole sent a blood-wave to the wrist, the pulse-rate was in reality only half as fast. It is essential, therefore, in every instance of suspected bradycardia that the heart be auscultated to determine whether there may not be apparent instead of actual bradycardia.

IV. STOKES-ADAMS DISEASE

By this term is designated a very remarkable and obscure complex of symptoms which consists in a paroxysmal intensification of an already existing bradycardia, together with vertigo or syncope and epileptiform seizures. Adams in 1827, and later Stokes, were the first to describe such attacks, and therefore Huchard devised the term now generally employed in commemoration of these two famous physicians. English and French clinicians were the ones chiefly who for many years paid special attention to this syndrome and reported cases. Within comparatively recent years, however, it has attracted the attention of German and American physicians, by whom numerous valuable contributions to the subject have been added. In America, Prentis and Edes deserve mention, while in Germany the most notable and latest articles with reports of cases are by His, Hoffman, and Jaquet. The exhaustive paper by the last named in Deutsches Arch. für klin. Med., Band lxxii, is particularly worthy of note.

Adams's original case was in a man of sixty-eight, and Stokes's 2 were in men of fifty-six and sixty-eight respectively, all 3 of whom presented clinical and post-mortem evidences of cardiac and vascular degeneration. It was thought, therefore, that the disease was limited to individuals well on in years. For instance, of 21 cases collected by Boyer from the literature there were only 2 whose age fell below fifty years. It is now known, however, that much younger persons may be befallen, and Jaquet states that he has found in literature 15 cases in which the age was below forty, and including his own, 9 that were below thirty years of age.

The **etiology** and **pathology** of this disease, if disease it is to be called, are still obscure, and hence a subject for speculation. Thus Stokes and early writers considered the attacks due to degeneration of the heart, Charcot to disease of the medulla, while Huchard and his pupils regarded it as the result of arteriosclerosis, especially of the coronary arteries. In Halberton's case the symptoms dated from a fall which injured the back of the head, and led, as shown by the autopsy, to great narrowing of the occipital foramen and consequent compression of the medulla. The disease has been attributed to lesions of the vagus and cardiac plexus, and in a few instances such structural lesions have been discovered at the necropsy.

On the other hand, fatal cases have been observed in which searching post-mortem investigation has failed to reveal any lesion capable of causing the symptoms, and indeed any recognisable changes that could be held responsible for the death of the patients. Such, indeed, was the state of things in Jaquet's case.

Hoffman's patient was a woman of twenty-three without clinical signs of cardiac or other organic disease, but with a severe anæmia. In her case the symptoms appeared to yield to inhalations of oxygen and other treatment appropriate to the blood-state. In other instances the disease has seemed to depend upon syphilis, indiscretions in diet, disorders of digestion, or obstinate constipation, the cure of which has favourably influenced the attacks. Jaquet's was an example of this kind, with a suspicion of syphilitic infection years before, and suitable treatment was instituted, together with correction of the constipation.

Syphilis appears to have had an etiological connection with the attacks in 5 cases (Jaquet). Emotional excitement, as a fit of anger, has been known to call forth a seizure, and did so in Jaquet's patient.

Tripier held that the disease was a genuine epilepsy, a view to which some features of the attacks in certain cases appear to lend support. It is now not so regarded, however, by the great majority of writers.

It is plain therefore that the pathogenesis is uncertain, but that we must now recognise two great groups, (1) in which the age of the individual is advanced and there are structural changes of the heart or vascular system, or definite lesions in the central or peripheral nervous system; (2) cases occurring in younger persons sometimes with clinical evidence of cardiac disease, sometimes without any demonstrable lesions either before or after death, and which appear to depend upon some obscure disturbance of the nervous system, as the brain, or upon an interference with normal cardiac contractions.

The former hypothesis is advanced by Jaquet, who thinks that the phenomena depend upon a cramp-like constriction of the vessels of the brain.

Hoffman, on the other hand, ascribes the symptoms to an interference with the ability of the cardiac muscle-fibres to respond to irritation or to conduct the impulses to contraction from the auricles to the ventricles. In other words, there is a block in the fibres that carry the impulses to contraction from the auricles to the ventricles.

From the foregoing, it is plain that we must enlarge our conception of this Stokes-Adams syndrome and not confine it to cases showing age or signs of cardiac or vascular disease, as was once done.

The **symptoms** which make up this singular clinical picture are, in the order of their frequency, (1) bradycardia and other phenomena connected with the circulatory system, (2) vertigo and syncopal attacks, (3) epileptiform convulsions, and (4) disorders of respiration. The first three are by far the most common, and in cases displaying all of them it is not always possible to determine which ushers in the series. It seems to be generally held, however, that the peculiarity of the heart's action soon to be described is the first to appear.

The pulse is habitually slow and usually regular in individuals displaying the complaint, but during the attack it becomes still slower, sinking to 20 or less, or, as in Halberton's case, to 5 in the minute. It may remain regular, or, as in my case (see page 324), may show marked irregularity in the intervals between the waves. Another peculiarity is its great tension, which in my patient was shown by Gaertner's tonometer to be 165 millimetres of mercury. Still another feature which I had observed, and upon which Jaquet comments, is the obstinacy with which the bradycardia is maintained in spite of diffusible stimulants and the effect of exercise. This persistence of the slow pulse-rate is observed of course during the intervals as well as in the attacks. When my patient's pulse is at 26 no amount of walking or even of stimulants is able to cause appreciable acceleration. Bax has reported an observation in a single case which was cited by Regnard—namely, that on one occasion the administration of digitalis sent up the pulse-rate to 60. Singularly enough a similar effect was reported by my patient to have followed his taking the same drug.

If during an attack the attending physician auscultates the præcordia, he in some cases hears nothing during the pause between the pulse-waves, while in other cases one or more extremely feeble cardiac tones, or possibly a faint systolic murmur, is audible. This peculiarity has been noted by many observers and by myself. These tones are as a rule not accompanied by perceptible impulse in the heart region, and for this and other reasons these feeble tones have been thought to indicate auricular contractions, but not ventricular, and hence are spoken of as abortive cardiac contractions. Such was the view held by Stokes, and recently maintained by Hoffman in his report of an interesting case in which he argues for this explanation at considerable length. Jaquet, on the contrary, has, for reasons that will be stated further on, come to the conclusion that they are in reality contractions of the ventricles.

Another phenomenon of great interest and singularity is sometimes perceived in the neck, and was first described by Stokes. This consists in feeble pulsations in the right internal jugular vein directly above the clavicle, and synchronous with the almost inaudible cardiac sounds. In Stokes's case there were two such "semi-beats," as he called them, between every two powerful cardiac systoles which sent a strong large pulse-wave into the carotids and peripheral arteries. Such was also the observation made by Jaquet, while in my patient these tiny jugular pulsations number two so long as the heart's action is regular, but, as will be found by reference to my case (page 327), may number many more during his attacks when the pulse is no longer slow and regular.

Jaquet, by recording a tracing of these jugular pulsations and a cardiogram simultaneously and afterward carefully measuring and interpreting the records thus obtained, came to the conclusion that these jugular waves do not indicate merely frustrated contractions of the right auricle, but are tokens of feeble systoles on the part of the right ventricle. He believes that owing to the enormous arterial tension, and hence peripheral resistance, the ventricles are not able to force open the semilunar valves in consequence of the feebleness of their (ventricular) contractions.

These are able, however, to drive a portion of the contents backward through the two auriculo-ventricular openings, and hence these jugular pulsations are venous waves caused by tricuspid insufficiency.

When at length the ventricular muscle has regained its ability to respond normally to the impulse to contraction discharged from the auricles, its systole is sufficiently energetic to overcome the resistance in the aorta and a blood-wave is sent along the arteries with a resulting pulse-wave. This either relieves the right ventricle and a jugular pulsation does not occur, or this latter is obscured by the massiveness of the pulsation in the carotid.

This explanation of Jaquet's is in accordance with what I have repeatedly observed in my patient—namely, that the feeble tones audible between every two loud cardiac sounds are synchronous with weak yet recognisable impulses of the heart against the chest-wall at the exact site of the powerful apex-beat. This it seems to me indicates that the ventricles contract feebly at these times, since I cannot conceive of auricular systoles being powerful enough to cause a perceptible though indistinct apex-beat.

It was this synchronism between the tiny waves in the neck and the feeble cardiac impulses which at first made me look upon the jugular pulsations as occurring in the common carotids and being too feeble to be transmitted as an appreciable wave higher up, beneath the angle of the jaw. Closer observation at a later date, however, convinced me that the pulsations were really in the internal jugular vein.

Another peculiarity in my patient, and which has been observed in other cases also, is that at times when he was wholly free from symptoms his heart beat 72 times a minute, and each beat was represented by a wave at the wrist. Moreover, the pulse was notably soft and a sphygmographic tracing taken at the time by Dr. E. F. Welles was commented on by this competent observer as showing striking want of capillary constriction, a condition in fact of capillary dilatation. Whereas whenever a sphygmogram was taken during this patient's state of pronounced bradycardia, it invariably manifested abnormal capillary resistance. These observations bear out Jaquet's contention that the attacks are due to abnormal vaso-motor constriction, or spasm in fact.

Vertigo is experienced to a greater or less degree in all cases, and annoyed my patient for over two years before more alarming phenomena set in. The assumption of the dorsal decubitus did not prevent its recurrence or even mitigate its severity. The dizziness was evidently a mild manifestation of cerebral anæmia that ultimately declared itself as distinct fainting fits.

Syncope may or may not be experienced, but in most cases is a prominent symptom. It does not last more than a few seconds or a minute as a rule, is ushered in by an ashen pallor, vanishes with a sudden rush of blood to the head, which produces an intense flush of the countenance and feeling of distention of the cerebrum that leaves a dull headache behind. A patient may have no recollection of what has transpired, or, as with mine, he may awake with a clear consciousness of his having fainted. During the moments of syncope the medical attendant perceives no pulse in any of the arteries, and may not even be able to detect any heart-sounds. Consequently, if the intermission persists for five or eight seconds he may be deceived into thinking death has really come.

Mild epileptiform convulsions are apt to be shown during the syncope. These may be no more than a twitching of the mouth, but they are generally observed in one or more of the extremities. In the case of my patient the convulsive movements are confined to the right arm and corners of the mouth. In most instances there is no foaming at the mouth, wounding of the tongue, or involuntary discharge of urine or fæces. Yet biting of the tongue has been noted, and in a case reported by His there was involuntary evacuation of the contents of the bladder. Huchard, I believe, was the first to direct attention to the association of epileptoid seizures with the bradycardia and vertigo previously noted. In the case observed by me there have never been involuntary discharges or any other more pronounced symptoms of epilepsy than the slight muscular contractions already mentioned.

Disturbance of respiration has been given as the fourth symptom of this singular group. It may occur, but is not at all common, and is said to consist of Cheyne-Stokes breathing, that may occur at apparently any time during the attack. It was observed by His, I believe.

Finally, this symptom-complex may occur at long or short intervals, but as a rule not daily or many times a day. Yet my patient has been known to have attacks as often as every five minutes for an hour or longer. They would then disappear for several hours or a day, after which they recurred with previous severity. This state of things had persisted for three weeks prior to his coming to Chicago and placing himself under my care.

It will be seen by referring to the more detailed report of his case in the chapter on Aortic Stenosis (page 324), that after this young man had put himself on a rigid vegetarian diet and had thereby regulated his bowels, he enjoyed immunity from his vertigo and intermittent pulse for a period of five months. He then returned to his former mode of diet, and had two premonitions of a return of his former symptoms. It was, however, quite a year before his attacks assumed the severity exhibited in March and April of 1902. At the present writing (June, 1902) this patient is enjoying a respite from his attacks, apparently as a result of treatment by which both digestion and the action of his bowels have been improved. His pulse is now running 26 to the minute with two incomplete systoles interposed between every two powerful cardiac contractions. The heart findings remain essentially as they were two years ago.

Notwithstanding the fact that patients who presented a cardiac murmur during life, have yet, after death, in a Stokes-Adams attack, been found entirely free from evidence of heart-disease, it is still my belief that in this case there is moderate obstruction at the aortic orifice. It is conceivable that this obstruction, acting in conjunction with the peripheral resistance of abnormally high blood-pressure, proves too much for the left ventricle, and thus leads to relative insufficiency of both sets of auriculo-ventricular valves, with consequent generation of the apex systolic murmur which has puzzled so many competent observers.

The cardiac defect in this case may possess some etiological connection with the attacks, but it has always been my opinion that the relation is an accidental one.

The condition which determines his attacks appears to reside in the intestinal tract, as shown by the results of diet, treatment, and numerous urine analyses. Urine passed by this young man during the days in which attacks occurred always showed a reduction of solids to below normal, and a quantitative estimation of the indican showed this substance to be within normal limits. So soon, however, as there came a period of several days entirely free from Stokes-Adams symptoms urinary solids increased, and in particular the indican rose to three and even four times the amount previously obtained. The accuracy of this observation was proved over and over again.

This, as it seems to me, may be taken to indicate a condition of auto-toxæmia which in some way excites the attacks. At all events, it is in line with Jaquet's observation in his case, in which the seizures disappeared for many months after constipation and indigestion had been removed. As in the case of my patient, they recurred with a return of digestive disorder and proved fatal in the fourth attack.

The *diagnosis* of Stokes-Adams disease presents difficulty when the paroxysms are characterized only by vertigo and increase of an already existing bradycardia. I did not recognise the real significance of my patient's symptoms until months had elapsed. There ought not to be any difficulty even in such unpronounced forms, provided one is on the lookout for them. They are not common, and hence the physician is not prepared to recognise them.

When syncope and epileptiform convulsions are also present there ought to be very little difficulty in determining the real nature of the case. The occurrence of brief periods of unconsciousness, during which the individual may fall to the ground, and the previously slow pulse grows still slower, and the discovery in such a person of stiff arteries or signs of cardiac disease, make up a group of symptoms that are sufficient to stamp the attack as one of Stokes-Adams disease.

This is especially true of an individual past middle age: but if this complex of symptoms is found in a person under forty, or still more below thirty, then the muscular spasms are very likely to arouse a suspicion of epilepsy. Indeed, my patient's attacks are so regarded by one of my colleagues. There is certainly what may be called a symptomatic but not an idiopathic *petit mal*, but if one will carefully note the action and sounds of the heart during an attack, he will be likely to correctly interpret the phenomena. The pulse-rate of epileptics is commonly slow, but during their seizures it does not become so slow and disordered in rhythm as is the rule in Stokes-Adams disease. Moreover, it must be remembered that these latter patients do not as a rule bite the tongue or have involuntary discharges of urine and fæces.

Prognosis is exceedingly grave and uncertain. Hoffman's patient appears to have recovered, and many other cases have been reported without mention of a fatal issue, but such instances do not alter the fact of a liability to death when the heart stands still for so long a time as is occasionally observed. The prospect of recovery depends upon the nature of the cause, the possibility of its discovery, and its amenability to treatment.

The etiology is still veiled in obscurity, and moreover we are not yet sure that there may not be various predisposing if not exciting factors. It is quite possible also that the disease in the aged with structural changes of heart, vessels, or central nervous system, as the bulb, may not constitute a group separate and distinct from that form occurring in persons below the age of thirty.

Treatment of this affection is unsatisfactory, because in the present state of our knowledge it is mainly symptomatic. If gastro-intestinal derangement is suspected to be an exciting factor, it is of course to be corrected. Hoffman found the daily inhalation of oxygen beneficial in his anæmic patient, and together with proper diet this seems to have removed the attacks. The same agent was freely employed by my patient and at first appeared to do good, but subsequently was found to exert no influence.

One of Stokes's men was able to mitigate or arrest an impending attack by supporting himself on his hands and knees and allowing his head to hang low, the position favouring improved cerebral circulation. The device is so simple that it should be tried.

Theoretically, diffusible stimulants, as ammonia, camphor, ether injections, etc., ought to be of benefit by arousing the heart to more rapid action. I found them of no avail in the case under my observation. For the same reason, and because it acts as a vaso-dilator, nitroglycerin thrown under the skin ought to mitigate an attack, but in my hands this remedy has utterly failed. It may be used, however, and should be given several times if no effect is observed to follow the first injection.

My patient found the greatest comfort from a hypodermic of ¼ of a grain of morphine administered subcutaneously. The remedy did not appear to exert any controlling influence over his symptoms, but served to steady his nerve and promote sleep, which in his state of dread was certainly a boon. The unexpected effect which followed the administration of digitalis in Bax's case, and according to report on one occasion in mine, would seem to justify its trial. Nevertheless I must add that when this remedy was ordered by me for my patient it utterly failed of such effect.

In fact I did not find anything that seemed to positively influence the seizures themselves. Maintaining free elimination through kidneys and bowels appeared to keep them off, for so long as such treatment was persisted in this patient enjoyed an immunity from his symptoms. At the present writing he is well, but how long he will remain so is a matter of uncertainty.

CHAPTER XXV

ANGINA PECTORIS

Definition.—Attacks of intense pain in the region of the heart, with more or less disturbance of cardiac action, usually accompanied by a feeling of constriction of the chest and a sense of impending death.

History.—Although this form of heart-pain was not systematically described until the latter part of the eighteenth century, yet a graphic account of his own sufferings from this complaint was given by Seneca, and in 1707 Morgagni gave a clear description of a paroxysm in a case of aortic aneurysm. In February, 1768, Rougnon addressed a letter to M. Lorry which contained the description of the death of a certain Captain Charles, who, from the account given by Rougnon, appears to have suffered from attacks of angina pectoris. It was Heberden, however, who in July, 1768, first systematically described this formidable complaint and who gave it the name by which it is now universally recognised. The names of John Hunter, Edward Jenner, and Parry are also intimately associated with its early history, Hunter having experienced it in his own person, and having ultimately died in an attack.

Jenner in 1799 pointed out a definite connection between sclerosis of the coronary arteries and angina pectoris. He is said to have refrained from publishing his views at an earlier date out of consideration for his famous friend, John Hunter, who during his life had held contrary opinions concerning its etiology. Parry gave it the name of "syncope anginosa," for, although he recognised its connection with coronary disease, he considered the attacks due to paralysis of the heart.

From this time forward the literature of the profession teems with contributions on the subject and with divers theories concerning its pathogenesis. Most of the earlier writers attributed the complaint to morbid anatomical changes in the heart itself. In 1816 Kreysig definitely stated that it was *due to ischæmia of the myocardium* in consequence of defective blood-supply from sclerosis of the coronary arteries. In 1821 Reeder amplified the theory of cardiac ischæmia by asserting that this condition might proceed not only from ossification of the coronaries, but also from any other disease—i. e., as atheroma of the aorta—which might be capable of shutting off the blood-supply to the heart-muscle. This same theory was likewise espoused by Tiedemann in 1843, and in 1875 Germain Sée described the case of an elderly man who had suffered from anginal seizures and in whom after his sudden death the mouths of the coronary arteries were found almost obliterated by atheromatous plaques situated on the intima of the aorta. Throughout the balance of their course the coronary vessels were healthy. These few instances are sufficient to show in a general way the trend of opinion on the part of the supporters of the so-called anatomical theory.

In 1834, says Huchard, the theory of the purely nervous mechanism of the attacks was formally announced by Gintrac, although its neuralgic character had been previously asserted by Baumes in 1808, and others. Gintrac attributed the pain to irritation of the fibres of the cardiac plexus, and in 1863 Lancereaux published 3 cases in which the autopsy revealed inflammatory or other changes of this plexus.

There has been wide variance of opinion among French authors concerning the nerves implicated. Thus Laennec considered it an affection of the sympathetic system and called it neuralgia cordis. Bouillaud regarded it as an affection of the phrenic nerves, and Peter, while accepting this view as applicable to some cases, also believed there was a neuritis of the cardiac nerves. Trousseau termed it an epileptiform neuralgia.

In Germany also the subject was extensively discussed and received a variety of explanations.

Romberg considers it as a mere neuralgia of the cardiac plexus, a view not unlike that of Friedreich, who thought it due to hyperæsthesia of that plexus. The names of Bamberger, Traube, Nothnagel, Landois, Eulenberg, Guttmann, Leyden, Rosenbach, and many others are identified with the literature of this interesting subject. Landois, who in 1863 subjected the question to a critical study from a physiological standpoint, divided angina pectoris into four groups, as follows: (1) Cases caused by disturbance of the excito-motor or accelerator nerves of the heart; (2) those due to irritation of the cardiac branches of the vagus; (3) cases arising from reflex irritation of the abdominal viscera—" angina reflectoria "; (4) such as arise from vaso-motor disturbance in various parts of the body—" angina vaso-motoria." Eulenberg also contributed an elaborate article on this subject in Ziemssen's Cyclopædia of Medicine, vol. xiv.

Leyden considers the attack due to degenerative and inflammatory changes in the heart-muscle depending upon disease of the coronary arteries, which changes lead to impairment of the heart's function.

According to Rosenbach, some alteration in the contractions of the cardiac muscle takes place, which alteration may, but does not necessarily, lead to functional weakness. In consequence of this change, irritation is imparted to the sensory tract, and this stimulus sets free the various forms of pain and anxiety characteristic of stenocardia, in accordance with irritability of the sensory apparatus and the function of the respective nerves composing it. Painful sensations are more or less pronounced according to whether the sensory centres are or are not accustomed to the irritation to which they are subjected.

According to Rosenbach's view, this true heart-pain is an indication of the heart-muscle being less able than usual to accommodate itself to sudden change taking place in the performance of its work; or, in other words, its ability to respond to demands from without for a display of extra effort is impaired. Now and then, also, obstacles residing in the heart itself and capable of interfering with its perfect action may interrupt the performance of its regular work and in like manner give rise to the phenomena of angina pectoris.

During the century just ended many contributions to this interesting subject have appeared in England and America, of which the most noteworthy were by Latham, Gairdner, and Osler. The last mentioned, in his Angina Pectoris and Allied States, deals with the affection in a very complete and entertaining manner. The most exhaustive discussion of the subject, however, to which I have had access is that by Huchard. His historical *résumé* of the various theories is complete and shows painstaking research. His own view of the pathogenesis of this formidable complaint is highly suggestive and will be stated in the appropriate place.

Pathology and Etiology.—It should be clearly understood at the outset that angina pectoris is but a symptom and not an independent affection. It therefore can have no morbid anatomical characters peculiar to itself. Its pathology is obscure, and hence there have been, and are still, various theories to explain its nature and mode of production.

It may be stated in a general way that angina pectoris is divisible into two forms: one incurable and likely to terminate in death, the other curable and not likely to end fatally. Some authors, therefore, following Walshe's classification, speak of a true and a false angina. Osler makes this distinction, while Balfour, Gibson, and others consider such a division irrational and unscientific, on the ground that all pain is real, and that there can be no such thing as true pain and false pain. There can be no great objection to these terms if it is understood that they are employed for the sake of convenience, to distinguish grave cases from those that are not grave.

Huchard also classifies cases of angina which are purely neurotic and not likely to terminate fatally under the head of pseudo-angina, which he makes include the reflex and toxic forms. In certain cases, however, he believes that nicotine poisoning is capable of producing the fatal form of angina pectoris.

The confusion and obscurity which so long characterized the consideration of this subject, and which indeed may be said to prevail largely even now, arose from the attempt to make the same pathology responsible for all cases of pain that merit the term of angina pectoris. On the other hand, the recognition of two entirely diverse groups renders the subject clear and simple, so that we are able to get a tolerably definite notion of its pathology.

The angina which always carries with it the possibility of sudden death, and which therefore may be called true or grave, is usually associated with, and therefore thought to be dependent upon, structural disease of the heart. Various lesions have been found in fatal cases, but they are all of such a kind as to interfere with the blood-supply to the heart-muscle. Coronary sclerosis is the most frequent, but inasmuch as all cases of this disease are not attended with angina pectoris, it is evident that there must be something more than mere sclerosis of these arteries. Accordingly it has been determined that it is not so much the fact of disease of these vessels as it is that this disease must interfere with cardiac circulation if it is to give rise to attacks of angina pectoris.

Shutting off of the blood-supply to a limited portion of the myocardium—i. e., by thrombosis of terminal twigs or even of a branch of considerable size—does not apparently always occasion this pain. If, however, one main trunk, or, still more, if both trunks are occluded, or if their lumen is sufficiently narrowed without being actually obstructed, then attacks of angina are very likely to occur. Accordingly, a condition which is specially apt to result in anginal seizures is narrowing of the mouths of the coronary arteries by the sclerotic process, as has been repeatedly proved at the necropsy in cases of this terrible agony.

It is upon the evidence furnished by such discoveries that the theory has been reared of ischæmia of the heart-muscle being the essential pathological factor in the causation of the fatal form known as true or grave angina pectoris. In this variety pain is usually absent so long as the individual is at rest or is not making exertion that requires unwonted labour on the part of the heart. Under such conditions the circulation of blood within the myocardium is sufficient for its needs, but when some emotion or extra physical effort calls for unusual heart-work, the narrowed coronary arteries are incapable of supplying the organ with an additional volume of blood and the ischæmia is intensified for the time being. Thereupon an attack of angina pectoris is evoked. According to this hypothesis, the nerve-filaments or ganglia with which the myocardium is so richly supplied become irritated by this deprivation of required blood and send an impression through the cardiac plexus up to the centre, which then responds by discharging a sensation of pain along certain nerve-trunks connected with certain segments of the spinal cord. This will again be considered in greater detail.

The pain thus induced promptly causes a cessation of exertion and consequent lessening of the heart's work. As the organ resumes its accustomed tranquility its blood-supply proves again sufficient for its needs, irritation ceases, and with it at last goes the pain. Whether or not this is the actual *modus operandi* of the attack, the assumption that it depends upon a diminution of the blood-supply seems borne out by the nature of other cardiac and vascular lesions sometimes discovered in fatal cases of angina—i. e., aneurysm and atheroma of the ascending aorta, insufficiency of the aortic valves, and very rarely extreme degrees of aortic and mitral stenosis. In cases of angina that appear due to aortic sclerosis the mouths of the coronary arteries are very commonly found extremely reduced in size by reason of calcareous plaques on the surface of the intima or by calcareous thickening of the aortic wall, while the coats of the nutrient arteries are also generally invaded by the same sclerotic process. In cases of aneurysm the coronaries are narrowed either by the direct effect of the aneurysm or by associated atheroma. In the diseases just mentioned the changes must be of a kind to obstruct blood-flow into the coronary arteries if they are to occasion the agonizing symptom under consideration.

In aortic regurgitation adequate blood-pressure in the nutrient vessels is not maintained, and under conditions of extra effort relative ischæmia of the hypertrophied left ventricle is produced. Mitral and aortic stenosis prevent the discharge of a normal volume of blood into the aorta, and consequently hinder the adequate flushing of the coronary arteries. By some authors the influence of valvular defects in the causation of angina is denied without associated sclerosis of aorta or coronaries, and certainly angina pectoris is exceedingly rare in valvular disease.

Nevertheless, I have observed typical angina pectoris in one case of pronounced aortic stenosis that was, from the history and patient's age, presumably of rheumatic origin and in two instances of aortic insufficiency. In the first case no autopsy was obtained, but in one of the patients whose aortic incompetence was associated with angina and whose case was described in the chapter on aortic regurgitation, the pain was found to be clearly due to sclerotic narrowing of the coronaries. I am inclined to believe, therefore, that aortic incompetence of itself is not so likely to lead to angina as is stenosis.

Post-mortem observation proves undeniably that the nutrition of the heart-muscle may suffer seriously in cases of valve-disease without associated coronary sclerosis, and that such myocardial degeneration is particularly likely to be found in long-standing and extreme stenosis of the aortic orifice. This indicates that during a long period of time the demands on the energy of the heart outstripped its nutrition owing to the small supply of blood sent through the stenosed orifice into the aorta and coronary system.

If in such a state of things the supply of blood to the heart-muscle is still further diminished, although but temporarily, then it is possible for a paroxysm of angina pectoris to occur. The influences capable of determining such a temporary increase of cardiac ischæmia may be vaso-motor spasm, affecting the coronaries, as suggested by Powell, or the continuance of undue physical exertion after such effort has begun to overtax the heart. In extreme aortic stenosis cardiac overstrain is shown by still greater feebleness of the pulse, and when such is the case the coronaries are still more imperfectly flushed. There is temporarily a relative cardiac ischæmia which makes an attack of angina pectoris possible. Fortunately such attacks are rare, yet on the hypothesis of cardiac ischæmia as the cause of angina the conditions favourable to its production are present.

Finally, it should be stated that the degenerative changes discovered in the myocardium of persons who have died in a paroxysm of angina pectoris probably bear no direct etiological relationship to the pain. Were it not so, this formidable agony would be far more common than it really is. Such degenerations of the heart-muscle are to be looked upon as the result of the pathological condition in the coronaries or aorta which have led to the angina.

It now remains to consider how the morbid anatomical conditions just mentioned act in the pathogenesis of this obscure complaint. They are to be regarded as predisposing factors merely, which to be operative require some additional influence, since, if such were not the case, all persons in whose heart such changes are found after death ought to have suffered from angina pectoris. This we know is not the case. It is this consideration which has given rise to the various hypotheses propounded in explanation of the symptom-complex. The best known of the theories have been stated already in the history of the complaint and do not call for repetition. I would direct attention particularly to Rosenbach's and to that of cardiac ischæmia.

With reference to the latter, I think one should also bear in mind the suggestion of Sir Douglas Powell that oftentimes we are obliged to assume the possibility of *relative cardiac ischæmia* if we are to understand how some cases originate—i. e., the conditions under which they originate, and the beneficial influence of certain lines of medication. According to this hypothesis, vasomotor spasm may affect the coronary arteries and temporarily seriously diminish the flushing of the heart-muscle with arterialized blood. The irritation thus evoked is declared by pain and often by disordered cardiac action. When, upon administration of vaso-dilators or on cessation of arterial spasm from other influences, the coronaries become relaxed, and hence better flushed, irritation ceases.

Whatever is the exact condition, however, the generally accepted view is that there is abnormal and excessive cardiac irritation which initiates the paroxysm. This is Huchard's view at all events, and herein he appears to coincide with Rosenbach.

According to Huchard, the course of vents in cases of grave angina is as follows: The heart itself is the starting-point of the attack. From here the stimulus ascends by way of the sensory centres and finally reaches the medulla. Thence it is reflected along the intercostal nerves and brachial plexus as a manifestation of pain. The stimulus next reaches the vagus centre, and from here an inhibitory impulse is sent down to the heart, the original starting-place, and declares itself by slowed, and it may be intermittent action of the heart.

Such an inhibitory action explains the sense of constriction and of impending death, as well as the dilatation of the cardiac cavities sometimes noted.

In pseudo-angina, on the contrary, the point of departure of the irritation is an intercostal nerve or some other peripheral or visceral nerve, whence, as in true angina, it also passes to the medulla. From there an impulse is sent out *not along intercostal nerves and brachial plexus*, but is passed down along the vagus or accelerator nerves of the heart to this organ. According to the route thus selected, the action of the heart becomes either rapid or slow and otherwise disordered.

In these two forms, therefore, the original source of irritation is entirely different and the circuit is traversed in a contrary direction. To my mind this is a highly satisfactory explanation of the differences in their clinical phenomena. It likewise proves serviceable in making up diagnosis and prognosis and in deciding on the mode of management.

Leaving now the pathology of angina pectoris, which, however much we may speculate upon it, is undeniably obscure, we come to the consideration of the remaining predisposing causes as well as the influences which directly excite an attack.

Aside from the anatomical conditions already considered, we are at once impressed by the important part played by age. Angina pectoris of coronary origin is emphatically a complaint of individuals who have passed middle age and have entered their sixth or seventh decade. This is an age in which the effects of arteriosclerosis usually declare themselves, and yet angina of this origin is sometimes observed before the fiftieth or even the fortieth year. One or two instances are on record of its occurrence in children. Nevertheless such facts do not invalidate the correctness of the statements made concerning the importance of age.

Males are much more frequently affected than are females, and of men who are befallen it is a noteworthy fact that it is especially the well-to-do and well-fed. Regarding the influence of sex, Osler states that out of his 40 cases but 3 occurred in women. I have notes of 32 cases, and of these, 7 were in the female sex. Of 2 that came to autopsy, 1 was a woman of sixty-six with aortic and mitral stenosis, with sclerosis of the aorta and coronaries; while in the other, as already stated, there was coronary obstruction and aortic regurgitation, both of sclerotic origin. Of the remaining 5 cases, 1 was in an aged woman presenting well-marked signs of arteriosclerosis, a hypertrophied left ventricle, and a harsh systolic basic bruit. Another was a comparatively young female with aortic regurgitation of uncertain origin, but whose questionable habits always made me suspicious of the likelihood of syphilitic infection. The third was in the lady of forty-four with signs of pure aortic stenosis whose case has been already described in the chapter on Aortic Stenosis, and will be again alluded to in this. The fourth was in a woman of fifty with aortic regurgitation, due probably to arteriosclerosis, to judge from her history and the arterial thickening. The fifth case was that of a woman of fifty-six who was corpulent and had thickened arteries with a greatly hypertrophied and dilated heart. As regards this striking discrepancy between the two sexes, it may be stated that the greater prevalence of angina pectoris among elderly men is to be referred not so much to sex *per se* as to those conditions which are responsible for the greater frequency of arteriosclerosis in the male sex.

Heredity is also a predisposing factor, what was said under this head concerning coronary sclerosis and myocardial degeneration being also applicable to the symptom now considered. Instances are on record of this formidable complaint having been passed down through three generations. It is not at all uncommon for both father and son to suffer or even die from angina pectoris. The most notable instances of the kind occurred in the persons of Thomas Arnold, of Rugby, and his equally well-known son, Matthew Arnold.

Gout predisposes to arteriosclerosis, and therefore to angina pectoris. Syphilis, alcohol, and, according to Huchard, tobacco, are also predisposing causes. The last named may, however, be an exciting cause.

The *exciting causes* of true, or coronary angina, as Huchard calls it, are conditions that suddenly raise blood-pressure in the aortic system, or, according to the theory of cardiac ischæmia, require more work of the heart than it can perform in consequence of its diminished blood-supply. Foremost among these is muscular effort. The patient may have had no premonition of the malady, when, one day, perchance immediately after breakfast, he starts out for a walk and is arrested by an indescribable attack of præcordial pain. In other instances the first paroxysm is experienced upon the patient attempting to hurry to reach a car, or the like, and thereafter is repeated whenever he quickens his footsteps beyond his usual pace. Atmospheric conditions undoubtedly intensify the influence of exertion, for on a day when the wind is easterly and raw these patients find themselves unable to walk at even their accustomed gait; while in warm weather and on still days, or here in Chicago, even in the depth of winter when the air is dry and cold and the sun bright, these patients frequently find themselves able to get about with comparative comfort.

I have known patients who could walk with ease when the stomach was empty, yet who invariably experienced pain, of greater or less severity, whenever attempting to walk soon after breakfast. This is undoubtedly to be explained by the augmentation of arterial tension, produced by the presence of food in the stomach. Nevertheless the rise of blood-pressure thus occasioned is of itself not sufficient to evoke an attack, since it requires in addition physical exertion.

Emotional states, as anger, worry, or excitement from whatever cause, which accelerate and intensify cardiac action, are also capable of evoking an attack of angina pectoris. This was illustrated in the historic case of John Hunter, who was wont to say that " his life was in the hands of any rascal who chose to annoy or tease him." As a matter of fact, Hunter died during a paroxysm brought on by a fit of silent rage in consequence of having been flatly contradicted at a meeting of the Board of Governors of St. George's Hospital, October 16, 1793. His coronary arteries were found ossified and the aorta dilated. I knew a gentleman of this city, a great sufferer from ferocious attacks of angina for many years, who frequently declared that nothing was so bad for him as a fit of temper.

It is very remarkable how diverse are the effects of exertion in different persons, or in the same individual at different times. I recall the instance of a gentleman of fifty-two, who, by the way, had been an inveterate smoker of strong Havana cigars, and who could not walk out of an evening without suffering from his angina. Yet on one occasion he assisted in carrying a loaded bookcase up a flight of stairs without experiencing the slightest pain. This patient ultimately died suddenly. I have another gentleman under occasional observation who invariably experiences more or less pain of a true anginal character the first thing in the morning when he goes into the bath-room to dress. The taking of a cool bath is almost sure to evoke one of his seizures. Excitement and exertion combined are particularly apt to call forth an attack. For this reason coitus is especially injurious to some of these patients. The influence of cold in determining a paroxysm has been referred to, and is shown by the inability of patients to face a cold wind, particularly when damp as well as cold, and by the effect of a cold bath.

Distention of the abdominal viscera by flatus and the taking of a full meal are enumerated among the exciting causes by some authors, but in my experience these have been operative only in connection with other determining factors, as exertion. Tobacco is counted by Huchard as both an exciting and a predisposing cause, and he narrates instances of individuals who had abandoned smoking because of the attacks of angina it induced, and who upon returning to their former habit again found it promptly followed by the same unpleasant effect.

Gibson likewise speaks of tea and coffee in this connection, saying they also claim their victims. Regarding the angina due to tobacco, Huchard recognises three forms: (1) Functional or relatively benign form, resulting from spasm of the coronary arteries, without disease of the myocardium, and which is the "Angina spasmo-tabagique," and which is recovered from by giving up the use of tobacco. (2) An organic angina of a grave character, resulting from coronary sclerosis and which is not recovered from by the abandonment of the tobacco habit. This is the "Angina sclero-tabagique." (3) The form most benign of all results from digestive troubles produced by the tobacco habit (gastralgia, dilatation of the stomach, and insensibility of the mucous membrane). This is the "Angina gastro-tabagique." Strictly speaking, these all, with the possible exceptions of the second form, belong to the pseudo-angina.

In the group of cases known as false angina, and which, according to Huchard, belong to the peripheral or visceral neuralgias, and which will be considered in connection with cardiac neuroses, the exciting causes are not always easy of recognition, yet, as stated emphatically by Huchard, are never due to effort. Herein, therefore, lies the great distinction between true and false angina. Occasionally in true angina, the paroxysms occur at night, arousing the patient from sleep, but, according to Huchard, this does not invalidate the statement that coronary angina is evoked by effort, nor are these cases to be classed as pseudo, because coming on at night.

Their advent during sleep is referable to some condition which augments blood-pressure and acts in the same manner as does effort made by these individuals. Some of these conditions may be flatulent distention of the bowels, coldness of the air in the sleeping apartment, an uncomfortable position during sleep, or the recumbent posture itself, which is known to augment blood-pressure (Huchard).

Clinical History and Features of an Attack.—The sufferer from angina of coronary disease is most often an elderly man of about sixty years of age who has been engaged in mercantile, professional, or literary pursuits rather than in manual labour, and who often presents the appearance of well-preserved health. He not infrequently states that prior to his first attack of angina he never had any symptoms that made him think his heart was affected, and that were it not for this symptom he would still think his heart as sound as ever.

Questioned concerning the symptoms for which he consults the physician, he says he has a pain in the front of the chest, which he locates at the upper and middle portion, frequently putting his hand over the manubrium sterni. He describes this pain as coming on suddenly, usually during a walk, and becoming so intense as to compel him to stand still until it goes away, which it usually does in a few moments. Further inquiry brings out the fact that associated with the pain is a feeling of oppression or weight on the chest, and in some cases a sense of impending death. The essential features of an attack, therefore, or the characteristic triad of symptoms, as it may be called, are (1) a substernal pain, that is usually so severe as to be an indescribable agony, (2) a sense of great constriction of the chest, a feeling as if this were being crushed or squeezed together, and (3) a sense of speedy or impending dissolution.

The duration of a paroxysm is not long, generally not more than a few minutes, probably because the violence of the agony necessitates a cessation of the effort occasioning it, and with the removal of its exciting cause the attack subsides. Occasionally it persists for twenty minutes or more, and when it occurs in the middle of the night it is apt to last longer than do day attacks. Thus it is seen that the pain may come on either by day or by night, but as a rule and particularly in mild cases it is more likely to manifest itself during the waking hours and when the patient is exposed to some obviously exciting cause. Nocturnal seizures are apt to be more severe as well as of greater duration, because, according to Huchard, the rise of blood-pressure incident to the recumbent posture does not subside quickly even after the patient leaves his bed, whereas that due to effort or emotion yields promptly to the removal of the cause.

The first seizure has been known to prove fatal, and on the other hand attacks have recurred at varying intervals for five, ten, fifteen, and even twenty-five years. A single sharp paroxysm has been followed by years of immunity, and in other instances, after having been absent for a long period, the malady has then assumed a frequently recurring type. Huchard speaks of the paroxysms as sometimes occurring with such frequency as to overlap each other, and thus become practically continuous with only remissions in severity, a condition which he terms *l'état de mal angineux*. Some patients are aware so soon as they arise in the morning that they are going to have a " bad day," as they say, or that they will have to be more than usually cautious lest they precipitate an attack. As a rule, however, anginal seizures come on abruptly without warning and with such agonizing intensity that the sufferer is compelled to stop in his tracks and remain standing, scarcely daring to stir or breathe lest he intensify his pain beyond all possibility of endurance.

At other times he leans against a tree or wall for support, or he stands in some peculiar attitude which experience has taught him will somewhat mitigate the severity of the pain. He may lean forward or bend backward, let his arms hang motionless at his side or stretch them above his head in an effort to fix the pectoral muscles so as to thereby increase the expansion of his chest, which seems to him to be compressed and too small for its contents. But whatever his attitude, it is, according to Huchard, always an upright rather than a recumbent position, which latter, by augmenting arterial tension, increases the severity of the attack.

Most happily for the patient the angina usually departs as quickly as it comes, and unless the attack has been one of unusual length or severity the victim feels as well immediately after as he did before the seizure. He is generally able to resume his walk, although perhaps rather more cautiously than before. Such is the history of a comparatively mild angina pectoris, but in some sad cases the attacks grow more frequent and more agonizing until at length the patient is not able to move in bed or engage in conversation without frightful suffering, and life becomes a miserable existence.

It now becomes necessary to consider the features of anginal attacks in detail. It has been stated that the pain is substernal; that is, that its seat of maximum intensity or its point of departure is beneath the upper or middle third of the breastbone. It is for this reason that Baumes applied to the complaint the name of sternalgia. The pain may remain centred beneath the sternum, but in most instances it radiates into the side of the thorax and along the course of the brachial plexus into the left shoulder or down the corresponding arm to the elbow or still farther, as far as the wrist, or even into the two fingers supplied by the ulnar nerve. In some cases the pain takes origin in the region of the apex-beat or epigastrium, or, as in the case of one of my patients, just above the ensiform appendix, whence it shoots into the left shoulder and down the left arm.

In rare instances the attack starts in the arm, at the wrist or elbow, and thence passes into the chest to the region of the heart. In one of Trousseau's patients the paroxysm began in the back of the neck, and then darted forward into the tongue and front of the thorax. Very exceptionally the pain may take origin in the left interscapular region or at the adjacent dorsal spines. But whatever is its point of departure the anguish radiates more or less widely throughout the chest, flashing from the cardiac area into the left, sometimes the right arm, and in some cases into both arms, or upward into the side of the neck or the occiput, or, instead, downward into the left half of the abdomen, and now and then even to the thigh. These latter lines of radiation are, however, very uncommon as compared with its course into the left shoulder and arm.

Very diverse terms are employed by patients in attempts to describe their agony. It is spoken of as crushing, grinding, tearing, cutting, burning, scalding, or, in want of appropriate adjectives, as indescribable, frightful, and the like. One of my patients, a lady with extreme aortic stenosis, depicted her anguish as "a feeling as if my chest were being crushed beneath the wheels of a passing train."

The sensation of pain is sometimes lost in the terrible distress occasioned by the sensation of the chest being squeezed in a vise, or of the walls being forced together from before backward.

Balfour describes it as if the heart were being "grasped by a mailed hand."

Then, as if this were not enough, the sensation of impending death is added, to complete this awful suffering. The lady just mentioned, said in reply to a query upon this point: "Oh, yes! of course I feel as though I were going to die, but I have learned by experience that I do not die, and therefore I no longer speak about it. I always used to declare that I knew I was going to die." Nearly all sufferers from severe angina pectoris agree in the assertion that no other pain can compare with the awfulness of its agony, and if it were not happily of short duration, life could not endure.

The physician is not often a witness of the terrible agony, the attack being over before he arrives, or his attention is so occupied in efforts for the patient's relief that he cannot note the several features. Nevertheless, from such observations as have been recorded, we possess certain facts concerning objective symptoms. The face is expressive of unspeakable agony; it is anxious and usually pale, and bedewed with perspiration, but it may be congested. The patient is *usually motionless during the height of the paroxysm*, yet it may be ushered in and terminated by restlessness. The extremities are usually cold, and the pulse is variable. It is sometimes stated to be unchanged, but is for the most part small and tense.

It may be regular or irregular and, if accelerated in the beginning, is likely to become slower than normal before the cessation of the pain. The size and rate of the pulse have given rise to much discussion, but the consensus of opinion seems to be that it is small, sharing in the condition of spasm, and that its rate is slow, indicating vagus stimulation. The heart sounds are usually clear, but feeble, although in some instances a systolic apex-murmur has been audible.

As already stated, the seizure usually subsides suddenly, leaving nothing more than a feeling of weakness behind. The pain in the upper extremities may be accompanied but is more often followed by a feeling of numbness, even by transient paresis. The lady to whom reference has been repeatedly made, said her left arm was always helpless after cessation of the suffering.

In the case of my aged female patient, already mentioned as having arteriosclerosis, the face was flushed during the attack, and the cessation of pain was followed by vomiting. I have stated that patients remain motionless during the paroxysms, yet I have known two instances, both men, who thought they obtained some relief by walking gently about the room while the pain was on. One of them was a well-known attorney, in whom the necropsy verified the diagnosis of coronary sclerosis and fatty degeneration of the left ventricle. In the other case the age of the patient, the thickened peripheral arteries, and the history of the attacks, left no doubt as to the nature of his angina. Douglas Powell states that when relief is produced by quiet walking it has seemed to him to indicate a fairly sound state of the heart-muscle. This may be so in some but not all cases. Mr. H., the attorney, was found to have extensive myocardial degeneration, and hence some other explanation is needed in his case. In my other patient, the gentleman was not only able to endure without pain certain gymnastic and breathing exercises which produced great perspiration, but he declared that he felt better for them. That they did not call forth his suffering the same as did walking against a wind, may have been due to the lowering of arterial tension which they induced.

The extremely variable course of the malady and its not infrequent termination in death during an attack are also noteworthy features. An historic instance is that of the Rev. Dr. Chalmers, who is thought to have died during a paroxysm of angina, since he was found dead in his bed with a bowl beside him into which he had emptied the contents of his stomach. I knew a similar instance of an old gentleman who, after having suffered attacks of angina pectoris for twenty years, was found lifeless in his bed in a hotel with a wash-bowl resting on the bed in front of him and containing vomited matter. I knew of another elderly gentleman who, while in attendance upon a lawn fête, was seized with a paroxysm of præcordial pain, vomited, and immediately died before assistance could reach him. So far as could be ascertained, it was his first and only attack.

Before leaving this subject, it should be mentioned that Gairdner has described what he calls *angina sine dolore.* What he means by the term is best described in his own words: "Apart from what has been variously termed cardiac asthma, dyspnœa, or orthopnœa, which in many cases receives its clear explanation from the associated states, either of the pulmonary circulation or of the lungs, bronchi, and pleuræ, as disclosed by physical signs, there is often an element of subjective abnormal sensation present in cardiac diseases, which, when it is not localized through the coincidence of pain, is a specially indefinable and indescribable sensation, almost always felt to be such by the patient himself. I make this remark deliberately, as the result of experience, and well knowing it is liable to be brought into question in particular instances—that, in fact, a large part of what has been described under the titles given at the commencement of this paragraph has been inextricably confounded by systematic writers with the sensation, or group of sensations, to which I refer.

To this group of sensations, when not distinctly accompanied by local pain, I have, in various instances, given the name of *angina sine dolore*, recognising thereby what I believe to be its true diagnostic and pathological significance, and its alliance with the painful angina of Heberden; the pain in which, however, as we have already seen, is an exceedingly variable element, both in degree and in kind."

Diagnosis.—Two questions present themselves for answer in the diagnosis of this formidable complaint: First, is the attack of pain angina pectoris? and second, what is the pathological condition underlying the attack? In other words, is the paroxysm to be classed as coronary angina? or is it a disorder of the nervous system independent of any cardiac or vascular disease? In attempting to answer the first query, one should keep clearly in mind the fact that all præcordial or so-called heart-pains are not attacks of angina pectoris. Many of these pains are simple intercostal neuralgias, and although variously described as cutting, stabbing, tearing, shooting, darting, burning, smarting, or only as dull, sore, heavy, and the like, they lack two features essential to angina pectoris—namely, the feeling of the chest being crushed, and the sense of the near presence of death.

The fact that pain is felt in the region of the left nipple or that it radiates from that point into the left shoulder and arm does not warrant the conclusion that it is angina. Indeed, a pain that takes its point of departure at the sternal end of the fourth left interspace or in the fifth left interspace below the breast, whatever be its direction of radiation, is more likely to be an intercostal neuralgia, since the agonizing suffering meriting the name angina pectoris is most frequently substernal. Moreover, in cases of intercostal neuralgia there are usually well-defined tender spots corresponding to the points of origin of the pain. Another characteristic of these intercostal neuralgias is their coming like a sudden stab or thrust, and then leaving as quickly, the points where they appeared being sore to the touch. When, as in some instances, the pain is permanent or is continuous, with exacerbations and remissions, the very length of the attack stamps it as intercostal neuralgia and not angina pectoris.

Moreover, these pains are most frequently met with in anæmic, neurasthenic, or otherwise neurotic individuals, or such as present symptoms of gastric disorder, and although by no means limited to the female sex, they are more frequent in women than in men, and generally in such as have not yet reached the age at which vascular degenerations are to be expected. In most cases attention to the points just mentioned enables one to readily differentiate intercostal neuralgias from the true heart-pain of angina. It is far otherwise, however, with those attacks of præcordial pain which display the features of true angina pectoris, yet which in reality do not belong to that class.

In other words, is it Heberden's angina with its possibility of sudden death? or is the pain a pseudo-angina, and hence not of the same serious import? The answer to these queries is to be found in the consideration of the following points: (1) the age and sex of the individual, (2) the state of the arteries and heart, (3) the influence of effort in evoking a paroxysm.

Attacks of præcordial pain that occur in young persons, no matter how closely they resemble coronary angina, are presumably symptomatic of irritation in some other organ than the heart, and if such attacks are in women the presumption is the stronger that they are false angina. If, on the contrary, they occur at an age when vascular degeneration is common, they are much more likely to be of the grave kind, even though they occur in females.

The detection of stiff arteries or of signs of heart-disease is in favour of true angina, and yet pain of visceral disturbance may occur in women past forty with hypertrophied hearts, particularly at the menopause or in such as have suffered all their life from recurrence is likewise of evil import. On the contrary, the prognosis may be said to improve in proportion as the attacks become less severe and the intervals between them longer.

According to Powell's assumption, previously mentioned, the prognosis should be better when relief is afforded by slow walking, but the case I have cited of the attorney proves the contrary. Moreover, in the case of my other patient his condition grew steadily worse in spite of his ability to endure the exercises to which he resorted in the vain hope of improving his general health, since his attacks of pain became more frequent if not more intense.

Treatment.—This includes, first, measures addressed to the relief of the paroxysms, and second, the management of the patient's daily life during the intervals between the seizures, with a view if possible to lessening their frequency and severity. The treatment of the attacks has already been considered in the chapter dealing with myocardial diseases secondary to coronary sclerosis, but may be again discussed at this time at somewhat greater length.

Very many and divers remedies have been used either solely to relieve the pain, or to strengthen and regulate the heart's action, and are therefore either anodynes or stimulants. Inhalations of chloroform and ether, Hoffman's anodyne, aromatic spirits of ammonia, opiates, carminative draughts, such were the measures relied upon prior to the discovery and introduction by Richardson and Lauder Brunton of nitrite of amyl. Two medicaments which in the experience of the profession the world over have proved of the highest value in controlling the attacks of angina pectoris, and now universally employed, are the nitrites and opium. The action of the nitrite of sodium is too slow, and therefore we have recourse either to the inhalation of a few drops of nitrite of amyl, or to a minim of a 1-per-cent solution of nitroglycerin dropped on the tongue. If amyl nitrite is preferred, it should be carried about by the patient in the form of nitrite-of-amyl pearls, containing 3 to 5 minims each of the remedy. Kept in this way the drug does not lose strength. So soon as the patient perceives his pain a pearl is to be crushed in the handkerchief, or a few drops from a vial may be poured thereon and the fumes inhaled, or the sufferer may breathe them directly from the vial. The action of the remedy is usually very prompt, rarely failing to afford relief.

There is usually no danger attending its use; at the most, only a dull headache is produced. If nitroglycerin be preferred, it is most conveniently and usually administered in the form of a tablet triturate containing 1 minim of a 1-per-cent solution. If the tablet is dissolved on the tongue instead of being swallowed, its effect is more promptly induced. This is especially the case if the occasion for its use is soon after the taking of food. The remedy can also be dropped on the tongue or taken in a swallow of water when the solution is preferred, but this method is not only less convenient, but it necessitates the loss of valuable time, when seconds of agony seem like hours to the sufferer.

Abatement or cessation of the attack generally takes place in a few seconds; but should this not be the case a second or even a third tablet may be employed at intervals of two or three minutes. Special indications for one or another of these remedies are found in pallor of the countenance and a small and tense pulse, whether slow or accelerated, regular or irregular, and intermittent or not, and in other signs of arterial spasm. The nitrites are essentially vaso-dilators, and stimulate the heart only indirectly through their dilating influence on the arterioles. Through their action, the wiry, and it may be slow, pulse grows softer, fuller, and more rapid, while at the same time there may be felt some constriction of the throat and tense or throbbing headache, symptoms which to the patient are of small moment in comparison with the relief from his frightful agony.

It has generally been my observation that in elderly individuals with sclerosis of the temporal, and presumably therefore of the cerebral arteries, the head symptoms occasioned by the nitrites are far less pronounced than in younger persons whose vessels are less stiff, and hence more responsive to the action of the drug. When phenomena of vascular spasm are absent or when relief does not promptly follow the use of the nitrites, recourse would better be had to opium in some form. A method of administration that yields speedy results is indicated, and therefore it is best to give morphine hypodermically and in a dose that will suffice without repetition—e. g., $\frac{1}{4}$ or even $\frac{1}{2}$ of a grain.

The lady to whom reference has been repeatedly made was compelled to resort to both nitroglycerin and morphine, and in addition frequently took a teaspoonful of sulphuric ether in sweet-ened ice-water. Relief was not obtained until under their combined effect the pulse became full and bounding, and the skin, previously cold and perspiring, grew flushed and warm. In her case there was extreme aortic stenosis with, it may be, coronary sclerosis, and a more decided stimulation of the heart was required than was indirectly occasioned by nitroglycerin. Under the influence of the ether, cardiac contractions are both invigorated and quickened, so that the coronaries previously dilated by nitroglycerin receive a more adequate supply of blood.

In comparatively mild cases relief may sometimes be obtained by the administration of diffusible stimulants, as aromatic spirits of ammonia, Hoffman's anodyne, camphor, whisky, or brandy, and their effect is hastened by being taken in hot water. Elixir of valerianate of ammonia in teaspoonful doses is a particularly eligible preparation, and admirably meets the indications when rapid stimulation is required. Any one of these remedies may be administered directly following the nitroglycerin and will sometimes obviate the necessity for morphine, a consideration of some importance in elderly individuals who, as well known, are sometimes peculiarly sensitive to this drug.

In nocturnal attacks, which are apt to be severe and prolonged, it is often well to supplement the action of medicinal agents by the application of hot bottles to the extremities or by heat to the præcordium, the epigastrium, or between the shoulders. There is no indication during an attack for the use of digitalis and strychnine, for not only is their action too slow, but when arterial spasm is responsible for the paroxysm the former will do harm. Aconite and veratrum viride should never be employed at such a time.

During the intervals between attacks the daily life of the patient should be so regulated as to minimize if possible both the frequency and severity of his seizures. If the complaint has existed for some time the sufferer is likely to have learned by experience that moderation in the matter of exercise is absolutely indispensable to immunity from his attacks. Nevertheless it may be well to caution him against undue exposure during cold and inclement weather, or going about insufficiently clad, against carrying heavy hand baggage or parcels, against attempting to walk soon after a meal, hurrying to catch a train or street car, etc. He should be explicitly instructed to make use of surface transportation in preference to elevated roads, which have to be reached by long flights of stairs, since the inclination to hasten up the last few steps as the train is heard approaching is almost irresistible, and such a spurt may precipitate an attack. Patients should also be instructed concerning the harmfulness of immoderate coitus, fits of passion, overeating, the too free indulgence in tobacco and alcoholic stimulants, of becoming excessively fatigued, etc.

The hands and feet should be kept warmly covered, and it is often well for these patients to wear a chest-protector both front and back. Those who can afford to pass the inclement seasons in a warm, equable climate should be advised to do so, since they can there take outdoor exercise without fear of encountering cold winds and of contracting attacks of bronchitis.

Sufferers from coronary angina have habitually high and sustained arterial tension, and as it is sudden and unexpected augmentation of this tension which often precipitates a paroxysm, it is essential that their blood-pressure be lowered. This can usually be accomplished, in a measure at least, by revision of the dietary —that is, by the restriction, or in some instances by the exclusion, of meats and the substitution of a largely vegetable dietary.

Rumpf, of Hamburg, interdicts the use of foods rich in lime-salts, as eggs, milk, cheese, spinach, etc. Theoretically, such a restriction is called for when there is arteriosclerosis, but practically, it will be found difficult to adequately nourish the patient if all foods rich in phosphates as well as meats are excluded. Furthermore, a too restricted dietary grows monotonous and leads to anorexia and feeble digestion.

The principles laid down for the dietary of cases of myocardial degeneration are equally applicable to these patients, and therefore the reader is referred to that chapter for details. Should arterial tension be not sufficiently reduced by regulation of the diet, then attempts must be made to accomplish this in other ways. To this end appropriate doses of nitroglycerin may be given every two or three hours during the day, or moderate doses of an iodide salt, three times daily, may accomplish the result. That such is the effect of iodine internally is generally held, and yet Romberg asserts that both clinical observation and experiment show this not to be the case. In some cases it may not be necessary to give nitroglycerin daily, but only on those days when the patient finds walking particularly difficult, or there is a raw easterly wind. I have known striking amelioration of the patient's condition follow regulation of the diet, together with the prolonged use of nitroglycerin and iodide of soda. Men addicted to the use of tobacco should be informed of its baneful effects and advised to abandon the habit altogether. If this is not acceded to, then the matter may be compromised by the patient's being allowed to smoke only mild domestic cigars. This will sometimes affect a cure of the tobacco habit in those who have been accustomed to choice Havanas.

In cases that have begun to manifest cardiac insufficiency or in which abnormally high blood-pressure threatens to soon overpower the heart, attempts must be made to restore cardiac strength or at least to stay its further decline. To this end recourse may be had to the usual heart-tonics. Strophanthus appears to me preferable to digitalis by reason of its inferior constricting effect on the arterioles, a virtue of the drug to which Frazer originally directed attention. If digitalis is selected, then its vaso-constrictor effect must be offset by the iodides or nitroglycerin. Strychnine and arsenious acid are also of benefit, and the former may be continued in moderate doses for many months. Strychnine is generally believed to raise pulse-tension, but this action is slight and not to be weighed in the balance as against its value as a heart-tonic.

The one method of treatment that is particularly adapted to this class of patients at this time are the so-called resistance exercises, and very favourable results have been reported from their employment in angina pectoris. Theoretical considerations, and indeed actual experience, indicate that benefit is also likely to follow the careful use of the saline baths with artificial as well as natural waters. Nevertheless, the lady whose case has been so often cited in these pages experienced her first severe paroxysm of angina pectoris shortly after her first bath at Bad Nauheim upon having been wheeled to her hotel, and then attempting to walk slowly from her wheel-chair to the elevator on her way to her apartments. Subsequent baths, however, were not followed by a similar distressful effect. Details concerning this mode of treatment are found elsewhere. (See chapter on Treatment of Valvular Disease in General.)

CHAPTER XXVI

SYPHILIS OF THE MYOCARDIUM—NEW GROWTHS IN THE MYOCARDIUM—ATROPHY OF THE HEART—SEGMENTATION AND FRAGMENTATION OF THE MYOCARDIUM

I. SYPHILIS OF THE MYOCARDIUM

Morbid Anatomy.—The most common myocardial manifestation of syphilitic infection consists in fatty degeneration of the cardiac muscle. This is not different in any way from fatty degeneration from other causes, and so is not recognisable except in the presence of other evidences of the disease. Associated with the arteriosclerosis of syphilis is a diffuse interstitial myocarditis, which is also usually classed as a luetic lesion. It seems probable, however, that in many cases the induration is due to the presence of the arterial disease, rather than to the direct action of the syphilitic poison.

Gumma of the heart is very rare, and especially so in the congenital form of the affection. The part of the heart most commonly affected is the wall of the left ventricle. The gummata appear as soft grayish masses surrounded by hyperplastic fibrous tissue, or if older, as dry caseous areas of a yellowish white colour. Very rarely a softening gumma may rupture into one of the cavities of the heart.

Etiology.—Syphilis attacks the myocardium only in the tertiary period of the disease, and after a lapse of five or ten years or longer following the initial sore. It is not confined to either sex, but appears to have been rather more frequently discovered in males. As regards age, it may be said to be more frequent at or after middle life, rarely in childhood for the reason that the disease is generally acquired, not congenital.

Symptoms.—Not only is heart-syphilis a comparatively rare affection, having been for the first time detected by Ricord, but its clinical recognition is still less frequent than is its post-mortem discovery. This is due to the fact of its possessing no pathognomonic features as yet recognised. Not only have patients, in whom this myocardial disease has been discovered after death, been known to exhibit no clinical evidence of heart-affection during life, but when symptoms were present they were found on analysis to differ in nowise from those displayed by persons suffering from other non-syphilitic forms of myocardial degeneration. Most observers agree in this statement that the cardiac action is likely to be disordered. This is generally though not invariably accelerated, and some authors, as Semmola, lay great stress on arrhythmia and acceleration of the pulse. Another symptom that has been noted is an indescribable præcordial distress which may or may not amount to actual pain. Philips has called attention to angina-like pain as having been present in one or two cases observed by him. This symptom was remarkably distressing on one occasion in a professional man, who subsequently died suddenly and in whom Philips found syphilis of the myocardium at the autopsy. Cardiac dyspnœa has also been complained of by some patients, but there was nothing about the difficulty of breathing that was in anywise peculiar.

Upon examination of the patient there may or may not be evidence of specific infection, such as old scars on the skin or mucous membranes, glandular induration, gummata, etc., and the arterial system may or may not furnish evidence of sclerosis. Physical examination of the heart is not infrequently negative, while in some cases there are signs of cardiac disease. When these are present, they are apt to be those of dilatation with feebleness or altered quality of the sounds. Murmurs are not present as a rule unless as an accidental complication or due to the dilatation—i. e., to relative insufficiency of the mitral valves, for example.

Diagnosis.—Unless there is a clear history of previous syphilitic infection the diagnosis of myocardial syphilis is not possible with certainty. On the other hand, even with such a history, one is not always justified in making the diagnosis merely because an individual of middle age displays cardiac symptoms. They may be due to changes in the heart-muscle incident to his age and not at all to syphilis. If one cannot discover syphilides of one sort or another, he should give the patient the benefit of the doubt until the futility of all other modes of treatment has been proved. The association of symptoms and signs of myocardial disease with a history and with clearly demonstrable lesions of the specific infection renders the existence of syphilis of the heart-wall very probable. If the cardiac manifestations occur in an individual not yet fifty years of age the supposition is greatly strengthened. Very often the diagnosis will have to be deferred until the results of specific treatment have been ascertained. Except by men of wide experience in this particular line of diseases the diagnosis of this cardiac affection must necessarily be a matter of guesswork in most instances. The clinical obscurity enveloping this affection is shown by the relative frequency with which it is found at the autopsy as compared with its *intra vitam* recognition.

Prognosis.—This may be said to be good provided the disease is recognised in time to institute proper treatment. In undiagnosed cases the prognosis is bad, since they are likely to terminate fatally. Death is apt to be sudden and unexpected. I know of no statistics going to show how long may be the duration of the disease, but it is probably a very chronic affection, having existed years, it may be, before the coming on of cardiac symptoms. The rapidity with which death is likely to follow the development of symptoms is likewise a matter of individual difference depending on the extent of the myocardial change, which is itself a matter we cannot obtain definite knowledge of during life. If the heart be extensively dilated, its action greatly disturbed, and the patient's symptoms pronounced, the prognosis is grave, and even specific treatment is not likely to do more than effect a partial recovery.

Treatment.—This, it needs hardly be stated, is the employment of iodides, with or without mercurials, as the physician determines. Being a tertiary manifestation, reliance is to be placed chiefly on the iodides. Ordinarily other remedies of the class of cardiac tonics are not necessary. But here again the medical adviser must decide. Their employment is symptomatic, and digitalis in conjunction with the specific medication may be of service in cases in which the action of the heart is much deranged and the patient's distress from dyspnœa is considerable. What has been said in other chapters on the hygienic management of heart patients applies equally to these, so long as cardiac power is deficient.

II. NEW GROWTHS IN THE MYOCARDIUM

Under this head are included various tumours and parasites. They are rare, some of them as parasites being excessively so, and aside from gummata just considered possess interest for the pathologist rather than the clinician. They will therefore receive only brief mention in this work.

Tubercles of the myocardium may be encountered as miliary nodules scattered through the heart-muscle, or still more rarely as caseous masses. The affection may also be declared as an interstitial myocarditis, which, however, possesses no distinctive features.

Parasites and cysts in this situation are still more infrequent and usually fail to declare their presence by either subjective or objective symptoms. Thus Knaggs, in the Lancet of 1896, vol. i, p. 29, narates the instance of a man who died suddenly, and had not previously manifested evidence of cardiac disease, yet in the wall of whose left ventricle a hydatid cyst was found at the necropsy.

Of other growths in the myocardium *cancer* is the most frequent, and yet this is absolutely very uncommon. *Lipoma* and *fibroma* have also been met with, but are still more rare. Malignant tumours occur in either the primary or secondary form, but of the two the latter is much the more frequent. The rarity of the primary form may be judged of by Gibson's statement that in 21,954 autopsies mentioned by Koehler, Tanchon, and Willigk there were only 21 instances of heart-cancer, while Petit found but 7 in the literature.

From Bodenheimer's analysis of 45 cases of secondary cancer, also cited by Gibson, it appears that the growth occurs most often as multiple nodules scattered throughout the myocardium, since it was limited to the wall of the left ventricle but seven times, to that of the right ventricle three times, and to the right auricle twice. It may occur at any age, even in infancy, but most often after forty-five, and is more frequent in males.

The clinical manifestations of myocardial cancer are too indefinite and uncertain to permit an *intra-vitam* diagnosis. The heart may be irregular and feeble in action, may furnish percussion evidence of dilatation, but in such findings there is nothing to distinguish these from ordinary cases of myocardial degeneration.

The prognosis is unfavourable, and yet for the most part life is destroyed in secondary cases by the original disease. In primary heart-cancer the tenure of life will depend largely upon the seat and nature of the tumour.

Treatment is of course purely symptomatic, since if the action of the heart is disordered and the real cause of the disorder is unsuspected or not, physicians find themselves limited to the administration of heart-tonics.

III. ATROPHY OF THE HEART

By atrophy of the heart is meant a diminution of the organ in weight and size. The condition may be partial or general. The former is exemplified in the smallness of the left ventricle seen in extreme mitral stenosis.

General atrophy may be the result of age, when it is spoken of as physiological, or the effect of disease—i. e., pathological. *Congenital smallness of the heart* is sometimes designated as atrophy, but, as preferred by Virchow, should be properly termed hypoplasia of the heart. It is usually associated with congenital smallness of the genitalia.

Morbid Anatomy.—The atrophied heart is of a brownish red or yellowish colour, often firmer than normal, sometimes presenting a wrinkled appearance, owing to puckering of the epicardium (like a withered pear, Eichhorst), and beneath the microscope the individual muscle-fibres are seen to be diminished in size, their transverse striation obscured and stained by a deposit of brown or yellow pigment near their nuclei. Adipose tissue is everywhere absent.

Etiology.—Various causes of general cardiac atrophy are enumerated, but those most often and powerfully operative are conditions which induce marasmus—i. e., pulmonary phthisis, cancer, diabetes, and chronic suppuration, as from disease of a bone. Thus W. Church is said to have obtained from the body of a woman who died of slow starvation in consequence of pylorus obstruction by carcinoma a heart that weighed only $3\frac{1}{61}$ ounces. Of 171 cases of phthisis analyzed by Quain the heart was atrophied in 54.4 per cent, while Engel is reported to have found cardiac atrophy in about 25 per cent of males who died of the same wasting disease between the ages of twenty-eight and thirty. It may here be stated that, according to Wunderlich, a heart is to be regarded as atrophied if it weighs less than 200 grammes.

Symptoms.—The clinical manifestations of atrophy of the myocardium are obscured by those of the general complaint, but may be said to be such as always characterize cardiac inadequacy —i. e., rapidity and weakness of the pulse, feebleness of cardiac impulse and sounds, without, however, signs of venous stasis other than slight œdema. As a matter of fact this œdema is due to malnutrition rather than to stasis.

Diagnosis.—The diagnosis is likewise obscured by the signs of the primary disease. It rests on the determination by percussion, or better by the fluoroscope, of marked decrease in the size of the heart, together with evidence of prolonged and extreme emaciation.

Prognosis.—The prognosis is that of the general cachexia, and yet a wasted heart may become so feeble as to cause death.

Treatment.—The treatment is that of the primary disorder, since it can do but little good to administer heart-tonics.

IV. SEGMENTATION AND FRAGMENTATION OF THE MYOCARDIUM

The precise nature of this condition has been, and still is, a matter of dispute. Opinion is still unsettled as regards its causation, the time of its occurrence, whether prior to or during the death agony, and consequently on the question whether or not it possesses any practical clinical importance. Renaut first described it as a segmentation of the heart-muscle due to chemical and nutritional changes and assigned to it definite clinical features. His original view was that the muscle-fibres became broken up, segmented, in consequence of softening of the cement substance holding the cells together. Various French and German writers, notably Przewoski and Klein and Browicz, confirmed Renaut's observations and indorsed his views. Others, chiefly von Recklinghausen and Tedeschi, discovered disintegration of the cardiac muscle-fibres, but declared it was due to rupture, i. e., fragmentation of the cells, which occurred during the death agony in consequence of overstimulation and irregular contractions.

Although they found fragmentation in otherwise normal hearts of individuals who had died suddenly by violence or otherwise, still in the majority of instances it was in hearts that showed chronic fibrous and fatty change, or the fragmentation was discovered in persons who had suffered from acute infections or lesions of the central nervous system. Indeed, Tedeschi found the condition in 48 per cent of 236 cases of death from all sorts of causes. The statements of von Recklinghausen caused Renaut to modify his views somewhat, and in 1894, at the first French Congress for Internal Medicine, he described the process as due to swelling, "gigantism" of the muscle-cells and alteration of the intercontractile plasma which render the cells brittle and disposed to fracture, while at the same time there is softening of the cement that leads to segmentation. Renaut still held, therefore, to his assertion that the process constitutes a distinct and recognisable clinical entity.

Since that time the subject has been discussed by numerous observers, chiefly in France and Germany. English and American writers have had little or nothing to say on the subject, because, it may be, of its being still *sub judice*, and as yet not believed to possess practical value to the clinician. The only important contributions that have, so far as I know, appeared in this country at this present writing, are by Ludwig Hektoen and John Bruce MacCallum. The former made a careful study of a large number of hearts from lower animals, both small and large, and from over 100 human beings that had died suddenly as a result of violence, or slowly or suddenly in consequence of a great variety of acute and chronic affections, some of them cases of either independent or secondary heart-disease. Hektoen's observations agreed with those of writers on the Continent as respects the frequency with which dissociation of the heart-muscle occurs in both sexes, at all ages, in all sorts of acute infectious and chronic diseases without associated cardiac lesions and in hearts manifesting the ordinary myocardial degenerations, hypertrophy and atrophy.

Thus, of 190 cases of deaths from a great variety of causes and in both sexes, he found segmentation in 65.78 per cent, while in 10 instances of traumatic and usually instantaneous death the condition was present in all. Hektoen states that whenever segmentation was present to any extent there was also more or less fragmentation. It is his opinion that segmentation is due to a disproportion between the violence of fibrillar contractions and the cohesive strength of the cement substance, and thinks that intravital alteration of the muscle-cells may predispose to cement-softening and consequent segmentation; it is not impossible, therefore, for excessive cardiac contractions during excitement, coitus, etc., to lead to sudden death through segmentation of the myocardium.

The symptoms attributed by Renaut to disintegration of the muscle-fibres are disordered action and feeble apex-impulse of the heart, some increase in the area of cardiac dulness, an uncertain systolic murmur, and it may be slight œdema. These are, however, not at all peculiar to segmented hearts, but are observed in hearts that have undergone other forms of degeneration. It is strange, therefore, that Renaut and his pupils should consider the process susceptible of clinical recognition. I shall not devote more space to its consideration, but allow the following sentences, taken from Hektoen's paper, to sum up the whole matter. "All the other authors regard general and focal segmentation as an accidental or secondary phenomenon occurring in the course of infections and intoxications in connection with the primary and secondary lesions of asystolic hearts, and with fatal traumatism. It constitutes an episode in the course of the principal affection. While it possesses an anatomical individuality, it is so common that it would be difficult to say in what disease it would surely be absent after, say, the twentieth year, and it would take a very long time to enumerate all the diseases in which it has been found present."

CHAPTER XXVII

PEDUNCULATED AND BALL-THROMBI OF THE HEART

Among the tumours of the heart may be included those rare formations which are found in the cardiac cavities and are in reality thrombi. They differ from cardiac thrombosis (marantic) in the chronicity of their development, the changes they undergo, and in their clinical history, since they do not give rise to emboli. Like vascular thrombi, some of them undergo organization, and when attached to the inner surface of the heart-wall by a pedicle are known as *pedunculated thrombi* or true polypi of the heart.

Others, called *ball-thrombi*, have either become detached from their pedicle, or having been formed by the deposition of successive layers of fibrin upon a primary nucleus, and unattached, roll about free in the chamber where they are formed. Both varieties are exceedingly rare, but of the two, ball-thrombi have been much less frequently encountered.

At the Reunion of Russian Physicians at St. Petersburg in 1893, in honour of Pirogoff, Pawlowski reported a case of true heart polypus that had come under his observation. In this paper he stated that diligent research in the literature up to that date had enabled him to collect only 25 cases, including his own. William Welch, however, in his admirable article on cardiac thrombosis in Allbutt's System of Medicine, states that he has found 8 others in the literature, making 33 in all. Small as is this number, that of *ball-thrombi* is still less. Von Ziemssen, in the report of a case at the Vienna meeting of the German Congress for Internal Medicine in 1890, stated that he had been able to collect only 4 cases besides his own. His research for published cases had been superficial, however, for Welch mentions 4 cases, with a reference to a fifth, that had been reported in England prior even to von Recklinghausen's, which by German authors was considered the earliest recorded. Since von Ziemssen's there have been others reported, so that up to date there have been 20 published instances of ball-thrombi. Some of these I had myself discovered in the literature before I had the good fortune to peruse Welch's article. The others have been taken from Welch's list. The entire number will be found at the close of this chapter.

Pedunculated thrombi may be found in any of the cardiac cavities excepting the right ventricle, although by far most frequently in the left auricle. Twenty-five were in this cavity, 4 in the right auricle, and a like number in the left ventricle. The point of attachment is various, although the interauricular sæptum seems to be the most frequent seat of the polypi, near the foramen ovale. Of Pawlowski's list of cases, 12 arose from the sæptum, 5 being from the fossa ovalis. Two, including Pawlowski's, were attached to the posterior wall of the left auricle, 2 within the appendix, and 1 to the mitral valve. In the other cases the precise point of attachment is not stated. In size and form the polypi differ, being likened to a pear, a small heart, a cone, a bullet, a walnut, and a hen's egg, the average comparison being to a walnut.

The pedicle is generally compact and strong, and in most cases the polyp is covered by a thin membrane thought to be an extension of the endocardium (Pawlowski). He also states that, according to Wilkinson King, some polypi could be injected through the coronary vessels, while in others this did not succeed. In some of the recorded cases the tumours contained calcareous deposits, others were cystic. In all instances of these heart-thrombi there is disease, usually narrowing, at the auriculo-ventricular orifice or some other condition, as dilatation, that has led to stagnation of the blood in the cardiac cavity containing the tumour. In Pawlowski's case there was mitral stenosis of an extreme degree.

Von Ziemssen states that *ball-thrombi* are for the most part of the size of a walnut, spherical, smooth, with no rounded corners, and showing no trace of a pedicle. In his case the mass was beautifully round and smooth, as if turned by machinery, and exhibited numerous indentations upon its surface. The thrombus was firm, and upon being sliced into sections showed successive layers of fibrin-formation. In the centre was a small mass that had evidently served as the basis upon which the fibrin had been deposited. Running up through the thrombus in radiating lines towards the circumference were delicate fibrous bands, which terminated each in a depression on the surface, and appeared by their organization and contraction to have occasioned the superficial indentations. The mitral orifice was also greatly stenosed in von Ziemssen's case. It may be remarked in passing that in his paper von Ziemssen alludes to his having had two other cases of pedunculated heart-thrombi, but Pawlowski does not include them in his list, and I have not discovered where they were published.

In Wood's case the ball measured 1½ inch in diameter, was of a dark-red colour, and made up of an outer wall ⅛ of an inch thick, composed of a large number of fibrinous laminæ and containing a mass of coagulated blood. The feature in this case, considered by Welch as unique, was that " adherent to the wall of the auricle, near the mitral valve, was a firm, oval thrombus on the free surface of which was a superficial concavity which formed a " kind of socket for the loose ball to roll in."

In one of Legg's cases, that of a woman brought into the hospital dead, two loose balls were discovered in the left auricle. In Osler's second case Welch states that an ovoid thrombus, resembling in size and shape a thick chestnut, was found with its smaller end sticking in the moderately narrowed funnel-shaped mitral orifice, from which it was readily removed. " At one pole of the thrombus was an irregular roughened spot indicating a former attachment, probably to a thrombus in the appendix."

In Arnold's case the ball-thrombus was elastic, as if composed of fluid incased by a thin membrane. At one spot the surface was roughened and of a speckled appearance, as if at this point it had once been in contact with the wall, while close by was a short thread-like prolongation which might have served as its means of attachment. The endocardium of the auricle was smooth and of normal appearance. The appendix was filled by a thrombus, broken down at its centre, and attached by a ribbon-like extremity to the internal aspect of the tip of the appendix. This mass projected into the cavity of the auricle. It is reasonable to infer, therefore, that these two thrombi were originally one, a small fragment having become detached and ultimately converted into the ball. The mitral orifice was the seat of obstructive disease.

In Redtenbacher's case there was a funnel-shaped mitral orifice and valve that barely admitted the tip of one finger. In the greatly dilated auricle were two thrombi, one a ball 3.5 centimetres in diameter, round, and even in contour, of a brownish-red colour, and covered with fine fibrous threads, soft and elastic; the other a long mass, which was attached inside the appendix by a pedicle, extended into the auricle.

From the very meagre description I have been able to find of Stange's case, it appears that a thrombus was found free in the interior of the left auricle, which thrombus was described as flattened (*abgeplatteten*). The mitral valves were slightly insufficient, and there was evidence of old aortic valvular disease. It may be questioned, therefore, if this case can be properly classified with von Ziemssen's and the others, since they all showed more or less stenosis of the auriculo-ventricular ring, and von Ziemssen expressly states that in typical instances mitral narrowing is present.

Ewart and Rolleston have described a cardiac thrombus which was discovered at the necropsy in a forty-three-year-old female. It was hour-glass in form, attached to the lower back part of the foramen ovale, and projected through the mitral orifice into the cavity of the left ventricle, but without disease of the ring or valve. The clot was old at its centre, with fresh fibrin deposits on its surface. The patient had had some chest trouble, probably pleuro-pneumonia, in February, 1896, and afterward a systolic apex-murmur with a snapping first sound; subsequently a presystolic bruit developed, and she died with symptoms of failing circulation from mitral disease.

This interesting case appears to be unique, since the orifice was not narrowed.

Pathogenesis and Etiology.—Two theories are offered to explain the formation of *pedunculated heart-thrombi.* One is that they are due to the coagulation of blood in the dilated cavity in consequence of the retardation of the stream incident to the obstruction at the auriculo-ventricular orifice. To this must also be added, according to von Recklinghausen's view of thrombosis in general, an eddying or whirling motion of the blood. These thrombi become attached to the wall, and subsequently undergo organization.

The other explanation is the one advanced by Bostroem, and accepted by both Welch and Romberg as applicable to some of the cardiac polypi at least. This is that true heart-polypi are thrombosed varices of small veins in the interauricular sæptum or result from hæmorrhages into the sæptum. This view is based on Bostroem's examination of two such polypi, one of which he showed to be a thrombosed varix, the other, which filled the right auricle, to be the result of hæmorrhage into the wall. " Therefore," says Welch, " it would appear that the nature of these formations is not always the same." It is this difference in the nature of heart-polypi which has led to the diversity of opinion concerning their origin.

Ball-thrombi are without doubt true heart-clots which may have been formed by the deposition of successive layers of fibrin probably upon a central nucleus or matrix. The question that does not appear to have been settled in respect to every reported case is whether they were formed as detached masses, or were originally parts of an attached coagulum, from which they had become broken off. Some of the balls have presented roughened spots and tiny rudimentary pedicles, which seemed to make it reasonably certain that they were once attached to thrombi discovered in the appendix. The smooth rounded form appears, as suggested by von Recklinghausen, to have been caused by their rolling about in the blood-stream.

Neither sex is exempt, yet women are more frequently befallen than are males, probably for the reason that they furnish a larger contingent of examples of mitral stenosis. Polypi have been found in the young and the old, yet, singularly enough, Pawlowski's list fails to comprise any case between the ages of twenty and thirty, a circumstance which he thinks may be utilized in arriving at a diagnosis. As regards ball-thrombi, however, there are several cases which were observed in persons of an age falling in this third decade of life. Finally, there must be a constrictive valvular disease to lead to stasis and coagulation of the blood.

Symptoms.—Whether the tumour is a pedunculated polypus or a ball-thrombus, the symptoms are such as characterize an extreme degree of circulatory embarrassment arising from stenosis of one or the other auriculo-ventricular orifice, generally the left. The patients usually suffer much from dyspnœa, even while at rest, the difficulty often assuming a paroxysmal or asthmatic type. Cough is present in most instances, and cyanosis is a noticeable feature. There is severe congestion of all the viscera, scanty albuminous urine, and œdema of the lower extremities, it may be of the serous cavities. The pulse may or may not be accelerated, but it is always strikingly small and feeble. Indeed, the scanty filling of the arterial system evinced by the pulse, and the exaggerated congestion in the veins, are features commented on by all observers. The almost total obliteration of the pulse is far in excess of what is observed even in high grades of mitral stenosis. In rhythm the pulse is not peculiar, since it may be irregular, intermittent, or unchanged.

A very striking symptom, which von Ziemssen lays stress upon as having been present in all three of his cases, was gangrene of a circumscribed area on the foot, associated with œdema and a truly cadaveric coldness of the extremities: phenomena due, in his opinion, not to embolism, but to arterial thrombosis. This results from the very deficient filling of the aortic system and sluggish flow in the arteries of the lower extremities.

In Pawlowski's case the patient, a female aged forty-seven, a school-teacher, the fatal illness lasted five weeks, and was characterized by an intermittent pyrexia, which at first gave rise to the diagnosis of typhoid. Great circulatory embarrassment and a mitral murmur did not at first attract attention, and indeed were variable, particularly the presystolic murmur. At the autopsy there was found in addition to the polypus and mitral obstruction a splenic tumour due to infarcts in its centre. These were broken down and purulent, and probably accounted for the septic fever.

One of Hertz's patients, a woman of thirty-nine, was admitted in a state of advanced cardiac feebleness and consequent circulatory embarrassment, and in spite of treatment died at the end of forty-eight hours. Arnold's patient was a servant-girl of twenty-three who entered the hospital with all appearances of some bronchial or pneumonic affection. A mitral lesion was discovered. Death took place four weeks after admission.

In Proust's case the patient was a man of fifty-eight who was ill five months with most distressing symptoms of embarrassed circulation, breathlessness, vertigo, cold sweats, and absence of pulse that were thought to depend upon mitral disease and secondary failure of the right ventricle. Death was the result of asphyxia. The necropsy disclosed a pedunculated thrombus in the right auricle 3 inches in length and attached to the sæptum.

It is thus seen that, however great may be the differences in the duration of the symptoms, these all evince a similarity in the manifestations of valvular obstruction of an extreme degree.

As regards the physical signs, these may be said to be those of a stenosis of an auriculo-ventricular orifice, usually the left. It must be remarked, however, that the characteristic presystolic murmur is not always present. Indeed, von Ziemssen states that after the thrombus has formed and begun to produce symptoms, the diastolic-presystolic murmur which previously existed may disappear. A very suggestive character of the murmur in such a case is its intermittency, coming and going, audible upon one examination and absent at another. This must depend in some way upon the presence of the mass, at one time the flow being sufficiently forcible to generate a bruit, at another too languid and small to produce sonorous vibrations.

Diagnosis.—This is obviously a matter of great difficulty if not of actual impossibility. So far as I can learn, an *intra-vitam* diagnosis has not been recorded. The existence of the thrombus must always be a matter of conjecture rather than certainty. However, if in a case of apparent mitral disease, or indeed of cardiac feebleness from any other cause, the embarrassment in the circulation be greater than seems accounted for by the lesion discovered, if localized gangrene of the foot occurs in a case of mitral disease, and evidently not due to arteriosclerosis or embolism, and lastly if a presystolic or other murmur comes and goes in an unaccountable fashion, one may entertain the suspicion of a heart-thrombus. One cannot from these data diagnose it with certainty.

Von Ziemssen considers three conditions indispensable to an *intra-vitam* diagnosis of an autochthonous cardiac thrombus: (1) There must be the physical signs of a mitral stenosis, since this lesion was present in all the typical cases on record. The evidence of this valvular defect must have been found at a time prior to the formation of the thrombus, however, because the murmur characteristic of stenosis disappears after the symptoms of thrombosis make their appearance. (2) Manifestations of an obstructive lesion of the left heart are not only indispensable, but they must be present to a degree not seen in simple stenosis. These are orthopnœa, cyanosis, coldness of the extremities, but, above all, *extraordinary smallness and feebleness* of the arterial circulation as evinced by the pulse. (3) The circumscribed gangrene of the foot which was present in all of his and one of Hertz's cases. With regard to this symptom, however, Redtenbacher calls attention to the fact of its absence in his case, although expressly stating that had the patient's life been sufficiently prolonged he believes it would have eventually resulted, such was the feebleness of the pulse.

Prognosis.—This is absolutely unfavourable, since the degree of obstruction to the circulation is incompatible with recovery of the patient or even with a tolerable existence after symptoms have once declared themselves. The exact mode of death is a matter of discussion. Hertz thought the ball-thrombus acted as a ball-valve and occasioned a total arrest of circulation by being driven into the orifice by the blood-current. Von Recklinghausen showed this to be unlikely, owing to the anatomical character of the stenosed opening. This is apt to present not a funnel-like cavity into which the ball might be pressed, but is a shallow depression of a transversely elliptical form so smooth as to favour the mass being rolled off again after once resting against the greatly contracted mitral opening. It is this supposed action present in Osler's case which has led to the appellation of " ball-valve " sometimes given to the condition. Death is likely to supervene, therefore, through strangulation or in consequence of cardiac or general asthenia, or through some of the immediate causes of dissolution, such as occur in severe valvular disease, or by reason of complications on the side of the lungs and general system. The fatal result is usually preceded by a longer or shorter period of suffering, and yet in Hartell's case the patient, a farmer aged fifty-nine, ate breakfast apparently in usual health, went to the field to work, and was found dead three quarters of an hour later.

Treatment.—This is purely symptomatic. Nothing can be done to remove the thrombus, even if its presence can be diagnos-

Pawlowski's List: Pedunculated Thrombus or True Polypus of the Heart

No.	Of the left auricle.	Of the right auricle.	Of the left ventricle.	Name of the authors.	Title of the journals.	Sex.	Age.	Place of attachment of the polypi.
		1809[1]	....	1. Burns.	Diseases of the Heart, p. 194.			
			1828[1]	2. Rigacci.	Arch. gen. de méd., xxvii, p. 276.			
		1832[2]	....	3. Zorrati.	Arch. gen. de méd., xxx, p. 401.			
1	1833	....	....	4. Choisy.	Revue méd., ii, p. 3.	M.	47	Sæptum.
2	1843	....	....	5. Dupuisaye.	Gaz. méd. de Paris, p. 270.	M.	19	Foramen ovale. Sæptum.
3	1843	....	....	6. Dubreuil.[1]	Gaz. méd. de Paris, p. 512.	M.	64	Foramen ovale. Sæptum.
4	1843	....	....	7. Dubreuil.[2]	Gaz. méd. de Paris, p. 512.	W.	34	Sæptum.
5	1845	....	....	8. Wilkinson King.[1]	Lancet, p. 428.	W.	55	Foramen ovale.
6	1845	....	....	9. Wilkinson King.[2]	Lancet.	M.	30	Auricle cord.
7	1845	....	....	10. Wilkinson King.[3]	Lancet.	W.	45	Foramen ovale.
8	1845	....	....	11. Wilkinson King.[4]	Lancet.	W.	18	
9	1850	....	....	12. Nuhn.	Schmidt's Jahrbücher, Bd. lxvi, p. 256.	....	..	Sæptum.
10	1853	....	....	13. Caron (Legendre).	Gaz. méd., 19 mai, and Bull. d. l. sc. an., 1854, p. 77.	W.	36	Sæptum.
		1863[2]	....	14. Ercole Galvagno.	Schmidt's Jahrbücher, 1871, and B. d. l. m. de bol., 1863.			
		1864[3]	....	15. Proust.	Compte rendu d. sc. m. et de biologie, i, 41.			
11	1868	....	....	16. Douglass.	Edinb. Med. Times.	M.	35	Back wall.
			1869[4]	17. Bucquoy.	Gaz. hebd., 5 mai.			
12	1872	....	....	18. Biermer.	Corresp. f. Schor. Aerzte, No. 9.	....	..	Sæptum.
13	1877	....	....	19. Millard.	Gaz. des hôpitaux, p. 534.	W.	30	Mitral valve.
14	1885	....	....	20. Hertz.[1]	Deut. Arch. f. klin. Med.. Bd. xxxvii, S. 74.	W.	39	Sæptum.
15	1885	....	....	21. Hertz.[2]	Deut. Arch. f. klin. Med., Bd. xxxvii, S. 74.	W.	74	Auricle cord.
16	1886	....	....	22. John Hartill.	Brit. Med. Journal, May 22d, p. 973.	M.	59	
17	1892	....	....	23. André Bergé.	Bull. d. l. s. an., v, s. 6, 11, p. 323.	W.	35	Sæptum.
18	1892	....	....	24. Veillon.	Bull. d. l. s. an., v, s. 6, 13, p. 361.	W.	76	Foramen ovale.
19	1893	....	....	25. Pawlowski.	Russkaja Medecina, No. 8 u. 9, 1894.			Back wall.
	19	4	2					

ticated. The associated valve-lesion will cause death eventually, and we can do no more than ameliorate the patient's distress. Indeed, we may deem ourselves fortunate if we can accomplish this.

BIBLIOGRAPHY OF CASES OF BALL-THROMBI

ARNOLD. Beiträge zur pathologischen Anatomie und zur allgemeinen Pathologie, Jena, 1890.
BOSTROEM. Deutsches Archiv für klin. Med., 1895, lv, p. 219.
EWART. Trans. Clin. Soc., London, 1896–'97, xxx, p. 190.
HARTILL. Brit. Med. Jour., May 22, 1886, p. 973.
HERTZ. Deutsches Archiv für klin. Med., Bd. xxxvii, S. 74.
OSLER. Johns Hopkins Hospital Reports, 1890, ii, p. 56. Montreal Med. Jour., 1897, xxv, p. 729.
PAWLOWSKI. Zeitschrift für klin. Med., 1894, xxvi, p. 482.
PROUST. Compte rendu d. sc. méd. et de biologie, 1864, i, p. 41.
RECKLINGHAUSEN, VON. Handbuch der allg. Path. des Kreislaufs u. d. Ernährung, 1883, p. 131.
REDTENBACHER. Wien. klin. Woch., 1892, v, p. 689.
ROLLESTON. Lancet, 1897, vi, p. 1546.
STANGE. Arb. a. d. path. Inst. in Göttingen, Berlin, 1893, S. 232–234.
WELCH. Allbutt's System of Medicine.
WOOD. Edinburgh Med. and Surg. Jour., 1814, x, p. 50.
ZIEMSSEN, VON. Vortrag gehalten auf dem IX. Congresse für inneren Med. in Wien, 1890, S. 281.

CHAPTER XXVIII

DEXTROCARDIA

This term signifies a transposition of the heart into the right side of the thorax. This condition may be congenital or acquired. Most congenital displacements of the heart occasionally met with possess interest chiefly for the pathologist. The organ may be situated in the cervical region, within the abdominal cavity or upon the exterior of the chest (ectopia cordis).

CONGENITAL DEXTROCARDIA

This form is the most frequent of all displacements and is of clinical as well as pathological interest, inasmuch as the physician may be called on to determine whether the displacement is pathological or normal to the individual concerned, and therefore devoid of danger. In most instances this abnormal situation of the heart is associated with transposition of the other viscera, a condition which has received the name *situs viscerum inversus.* That this is not invariable has been noticed by Breschet.

The displaced heart occupies the same relative position on the right side as it does normally at the left, while the stomach and spleen are in the right and the liver in the left hypochondrium. The position of the intestines is also reversed, so that the rectum lies in the right instead of in the left iliac fossa.

Symptoms.—*Congenital dextrocardia* occasions no symptoms unless it be associated with other cardiac anomalies, as sometimes is the case. It is stated, however, that patients with this displacement of the heart are apt to develop pulmonary tuberculosis. Apropos of this possibility I recall the case of a Miss A., who applied to me for an examination because she had had her attention directed to the fact that her heart pulsated upon her right side, and she desired to learn if it possessed any special importance. Examination showed the apex-shock was in the fifth right interspace, about 1 inch inside the vertical nipple-line. Cardiac dulness was of normal extent, and beginning a finger's breadth to the left of the sternum, reached nearly to the right mamillary line. The heart-sounds were of normal strength and clearness, and were situated at the right of the sternum. Percussion of the abdomen showed gastric tympany beneath the right costal arch and hepatic dulness in the left hypochondrium. At that time the patient was in perfect health and gave no history of tuberculosis in the family. Yet before two years had elapsed she developed pulmonary tuberculosis, to which she succumbed about a year later.

Diagnosis.—The detection of the dextrocardia depends upon the recognition of the cardiac impulse, dulness, and sounds to the right of the median line and their absence at the left. Its congenital nature is shown by the transposition of the abdominal viscera, which can scarcely be a matter of difficulty of determination.

ACQUIRED DEXTROCARDIA

Morbid Anatomy.—This form of dextrocardia may be complete, the heart lying entirely within the right half of the thorax, or it may be partial, in which case the organ is situated mainly but not wholly to the right of the median line. As this transposition of the heart is a pathological condition, the other viscera remain in their customary position. The morbid anatomical appearances in these cases are found chiefly in the lungs and their investing membranes, since the heart is not necessarily the seat of any other disease than that incident to the torsion of its supports.

The organ is fixed at its base by the great vessels, and cannot become displaced in either direction without undergoing more or less rotation upon its long axis. In dextrocardia there must be twisting of the arteries and veins at its base, and hence authors have speculated on the direction in which the heart must turn to admit of displacement to the right. Sibson maintained that the heart rotates in such manner as to bring the left ventricle to the front and the right chambers to the rear, while von Schroetter argued that the right ventricle turns towards the left so that the left ventricle recedes still further into the background.

A moment's reflection will convince one, however, that the direction in which the heart rotates is determined by the displacement and twisting of its supports or by the point of attachment of adhesions and the angle in which they pull. In a paper on dextrocardia, contributed by me in 1888, this question was fully discussed, and I there reported 2 cases which proved conclusively that the heart may rotate in either direction, so that both Sibson and von Schroetter were right. (For details see Medical News, 1884–1888.)

The twisting and strain to which the aorta and pulmonary artery are subjected may exert a detrimental effect on the heart. Thus in the case of a child which I reported the aorta was found constricted by the superior vena cava, which was stretched tightly across it, and the narrowing of the aorta thus occasioned had led to dilatation of the left ventricle. It is possible, therefore, for this abnormal and constrained position of the heart to lead to its hypertrophy and dilatation and to constriction as well as stretching of the large vessels at its base.

Etiology.—This is found in pathological processes that exert either pressure or traction upon the heart. The former is brought about through the accumulation in the left pleural cavity of air (pneumothorax) or of liquids (pleuritis with effusion and empyema). With the absorption or artificial removal of the exudation the heart usually returns to its normal situation, but the formation of pleuritic adhesions and obliteration of the left pleural sac may serve to maintain the organ in its acquired location. The pressure exerted may be sufficient to push the heart entirely beyond the median line, so that its apex strikes the chest-wall outside the right mamillary line, and Walshe says this may take place within thirty-six hours. Ordinarily the organ is not greatly displaced, and the apex may come to lie at any point between the midsternal line and the right nipple.

When the heart is drawn over into the right side, it is through the traction exerted by pleuro-pericardial adhesions acting in conjunction with more or less cirrhosis of the right lung. This was the cause in all three of my cases. The primary cause may be a trauma, or tuberculosis of the lung may be the initial etiological factor. Whatever be the predisposing cause, the pleuritic adhesions undergo contraction slowly, and a considerable length of time must elapse before the dextrocardia is completed. In this class of cases, moreover, are seen the most extreme examples of cardiac transposition, the heart assuming a nearly horizontal position in its new situation. It lies, of course, under these conditions, immediately beneath the anterior chest-wall and is uncovered by lung.

Symptoms.—These may consist of those phenomena ordinarily associated with venous stasis—i. e., cyanosis, dyspnœa, feebleness and rapidity of the pulse, palpitation, and after a time œdema, scantiness of the urine, and other evidences of visceral congestion, or the clinical picture may be rather that of the pulmonary affection with or without symptoms of cardiac insufficiency. The symptoms may be of a severe type throughout, but more frequently the course of the disease is protracted, and the symptoms are mild, depending upon the nature of the associated pulmonary affection. In a word, there is nothing distinctive of the clinical history of these cases unless it be their chronicity.

Diagnosis.—The detection of the fact of the dextrocardia can hardly be a matter of difficulty, particularly in cases in which it is associated with or dependent upon chronic disease of the right lung. When due to accumulation of air or liquid in the left pleural cavity with compensatory emphysema of the right lung, the condition may escape the detection of the careless observer. It is conceivable also that an aneurysm pulsating low down and to the right of the sternum, or a pulsating empyema between the sternum and right nipple, might mislead the inexperienced or superficial examiner. The history of the case and careful exploration of the chest ought, however, to protect against so gross an error.

Inspection and Palpation.—These disclose pulsation in the region of the right nipple and its absence in its usual situation.

Percussion.—This reveals an area of absolute and relative dulness to the right of the sternum having the characteristic outline of the heart, while a similar area of dulness is absent on the left. Unlike congenital cases, percussion discloses gastric tympany and hepatic dulness in their normal position.

Auscultation.—This enables one to perceive that, instead of the heart-sounds being audible in their normal situation, they are heard at the right of the median line.

The physical signs, by which are recognised the pulmonary diseases that bring about an acquired dextrocardia, do not need to be here stated.

If occasionally cardiac murmurs are heard in this class of cases, it is not always easy to determine whether they are organic from valvular disease, or are accidental and due in some way to the alterations in the cardiac walls and large vessels incident to the rotation of the organ. The history of cases of acquired dextrocardia shows that accidental bruits are not uncommon. For the differentiation of the murmurs one must rely on the rules that have been stated already in the introductory chapter.

Prognosis.—In most instances this may be said to be that of the lung condition, and yet in a case of complete acquired dextrocardia with presumably considerable torsion of the vessels, the condition is likely to shorten the prospect of the patient's life. Nevertheless, one of my patients was alive and in ordinary health fourteen years after my first examination. The prognosis in each case depends upon the evidence or not of cardiac feebleness and disordered circulation, all of which signs have been sufficiently set forth in previous chapters.

Treatment.—This must be based on the indications of each case and the principles that apply to other forms of cardiac inadequacy. It is needless to remark that nothing can be done for the relief of the dextrocardia in those instances in which it is owing to traction from permanent disease within the right half of the thorax.

CHAPTER XXIX

CONGENITAL DISEASES OF THE HEART

Some of these possess a pathological rather than a clinical interest, since they render extra-uterine existence impossible. For a detailed description of such the reader is referred to works on pathology. Congenital cardiac affections were the object of much interest and even of superstition in the early days of anatomic investigation. It is to Meckel, Bouillaud, Rokitansky, Dorsch, Peacock, Kussmaul, and Lebert that the profession is chiefly indebted for a scientific elucidation of their various modes of development.

Morbid Anatomy.—Of the congenital defects of the heart that are the result of developmental errors, the most frequently found and at the same time the least important clinically, is an increase in the number of cusps in the semilunar valves of the aorta or pulmonary artery. This condition is more frequent at the pulmonary than at the aortic opening. Four and even five segments have been found. The supernumerary cusps are usually smaller than the others, but the ring may be equally divided between the increased number of segments. The presence of a diminished number is of less frequent occurrence. Two cusps have then become united, leaving no trace of the line of union, or at best a very slight one. According to Osler, this condition is more common at the aortic orifice, but two of his twenty-one instances having occurred at the pulmonary. Osler further states that this defect is an important one, as the conjoined cusps are very apt to undergo sclerotic changes.

Stenosis of the pulmonary or aortic orifices may result from the more or less complete fusion of all three cusps (Fig. 78), and this may even proceed to complete atresia. The fusion may be the result of fœtal endocarditis or developmental error. In the former case the valve presents much the same appearance as after postnatal endocarditis. Vegetations may cover the cusps, project into the ventricle, or fill the sinuses of Valsalva. At other times, however, the united valves may present no signs of endocarditis, being combined to form a funnel, which may show signs of very slight sclerosis. Stenosis or atresia of the auriculo-ventricular orifices is of much less frequent occurrence than of the arterial openings. In either case the congenital disease is more frequent on the right side on account of the more frequent location of fœtal endocarditis on that side. Pott says that for one congenital aortic defect there are twenty-five pulmonary and tricuspid.

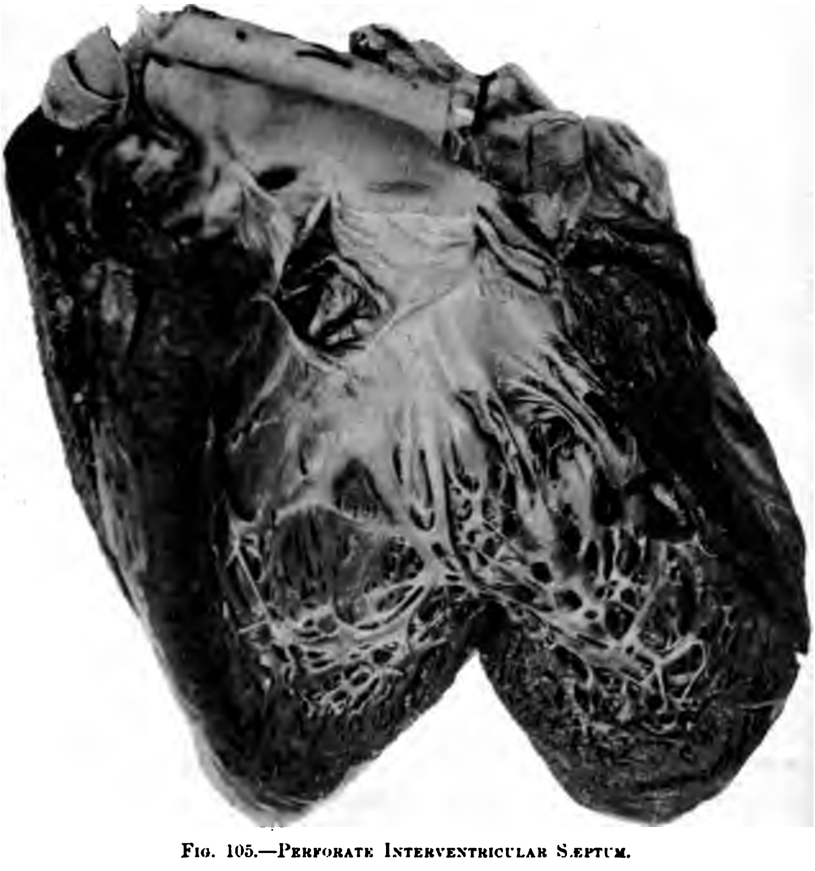


FIG. 105.—PERFORATE INTERVENTRICULAR SEPTUM.

Pulmonary stenosis, already considered in a special chapter, is a by no means infrequent congenital anomaly. Aortic obstruction is far less frequently congenital. In either case if the obstruction arises earlier than the eighth week of fœtal life, it leads to an imperfect formation of the interventricular sæptum. This is due to the inequality of blood-pressure in the two ventricles occasioned by the stenosis, and the consequent passage of a stream of blood from one to the other through the still imperfect sæptum, with each systole of the ventricles. This stream prevents the union of the two fundaments of the sæptum, and in consequence, the imperfection is almost always situated at the *pars membranacea*, or point where the two embryonic fundaments fuse (Fig. 105). This is high up on the sæptum in the portion separating the two *coni arteriosi*.

If the obstruction be established later in embryonic life, the interventricular sæptum is usually found entire, but the interauricular sæptum is usually imperfect, and the ductus arteriosus open. The stenosis need not necessarily be located at the valve to produce these effects, since narrowing of the conus on either side, the so-called stenosis of the heart, acts in the same way. It is not always possible to say whether the imperfect closure of the sæptum preceded the obstruction of the pulmonary ostium or of the conus, or whether it followed the other lesion. In the light of Küssmaul's conclusions, that defects of development predisposes to endocarditis, the former hypothesis is not unlikely.

Patency of the foramen ovale results from any condition causing a considerable inequality in the blood-pressure in the two auricles at the time when it is normally closed. This may be due to stenosis of one or the other of the auriculo-ventricular orifices, or obstruction at either of the arterial openings may secondarily influence the blood-pressure in the auricles, and so cause persistence of the foramen. The condition is often combined with a defective interventricular sæptum, or patent ductus arteriosus, for the reason that all these imperfections are due to the same cause. Patency of the foramen ovale, or rather an incomplete union of the valve with the ring, is by no means always to be considered a pathological condition. According to Romberg, such a condition exists in at least half of all cases. This may not produce symptoms, however, as when the valvular flap is of sufficient size the pressure of the blood in the left auricle keeps it closed and prevents any interchange of blood.

The ductus arteriosus persists as a patulous vessel, when, at the time it should normally be obliterated, the blood-pressure in the aorta and pulmonary artery is so unequal that a current flows through the ductus from one to the other. Thus in a case of pulmonary stenosis developing early in fœtal life, the contents of the right ventricle, experiencing difficulty in passing through the pulmonary orifice, enter the left chamber through the imperfect interventricular sæptum, and only a diminished quantity of blood passes into the pulmonary artery.

On the other hand, the aorta receives an increased amount of blood on account of the extra supply to the left ventricle from the right chamber through the imperfect sæptum. Thus the tension in the aorta is rendered higher than that in the pulmonary artery, and a portion of blood passes into the latter vessel through the ductus Botalli. The stream in the ductus, it is to be noted, is in this case flowing in a direction opposite to that normal in fœtal life, which is from the pulmonary artery into the aorta.

Persistence of the ductus may depend on aortic as well as pulmonary defect, and may be due to a congenital reduction of the calibre of the vessel, as in Fig. 107. The extreme case of atresia of either artery necessitates the patency of the ductus for the carrying on of the circulation.

Etiology.—There has been much speculation upon the determining factors in the development of congenital affections of the heart. Fœtal endocarditis is quite generally attributed to the agency of infectious diseases operating through the maternal circulation. It has not been at all clear what influences lead to the production of developmental anomalies. Some have sought to account for these in tendency or inclination to perversion of growth impressed upon the germ by the parent, and hence regard such abnormalities as stigmata of degeneracy.* This hypothesis is based largely on the fact that developmental defects of other parts of the body are not infrequently associated with congenital cardiac anomalies. Others, again, hold that these abnormalities, developmental as well as endocarditic, are the result of pathogenic agencies, the differences in result being determined by the period of fœtal life at which these agencies work. This conclusion appears justified by the results of Féré's experiments.

---

* F. Simpson, in 4,252 autopsies of the insane, found fenestration of the aortic valve 75 times; of the right semilunar, 18; of the mitral, 6; and of the tricuspid, 2. It was especially frequent in men. Supernumerary and rudimentary valves were found very often. It would be interesting to know how these findings would compare with those from the same number of necropsies of the sane.

This investigator found that if eggs in the stage of incubation were inoculated at a sufficiently early period with pathogenic organisms or their toxins errors of development resulted. This is certainly a very satisfactory explanation, and is one that accords with our modern notions of the bacterial origin of most maladies, It is more reasonable also than the assumption that defects in the septa are secondary to an inflammatory process that was limited to the orifice affected, since, as pertinently suggested by Osler, it is difficult to understand how an inflammation could fail to attack the whole heart at a time when the fœtus and heart are so diminutive.

The reason for the predominance of endocarditis in the right as compared with the left heart *in utero* is probably to be found in the greater blood-pressure within the right chambers. After birth has altered the course of the blood-stream by calling into use the vessels of the pulmonic system, blood-pressure becomes higher in the left heart, and this half now becomes relatively more liable to inflammatory processes.

Symptoms.—The disorders now under consideration do not possess individuality as regards their clinical features. Patency of the foramen ovale even when of considerable size does not necessarily preclude the possibility of long life and may not give rise to symptoms. Duroziez, cited by Gibson, discovered such a condition in a woman who died of erysipelas at the age of seventy-six.

When not dependent upon pulmonary stenosis or other valvular defect there may even be an absence of murmur or other objective evidence of the patency.

A defect in the interventricular sæptum may also fail to manifest itself by subjective symptoms, and there may not be even cyanosis, which, as we shall see later on, is ordinarily one of the commonest and most significant features of congenital heart-disease.

Stenosis or atresia of the pulmonary orifice or artery, on the other hand, rarely fails to occasion grave circulatory embarrassment, and hence well-marked subjective and objective symptoms. It is in this the most frequently recognised congenital affection, therefore, especially when attended by sæptum imperfections, that

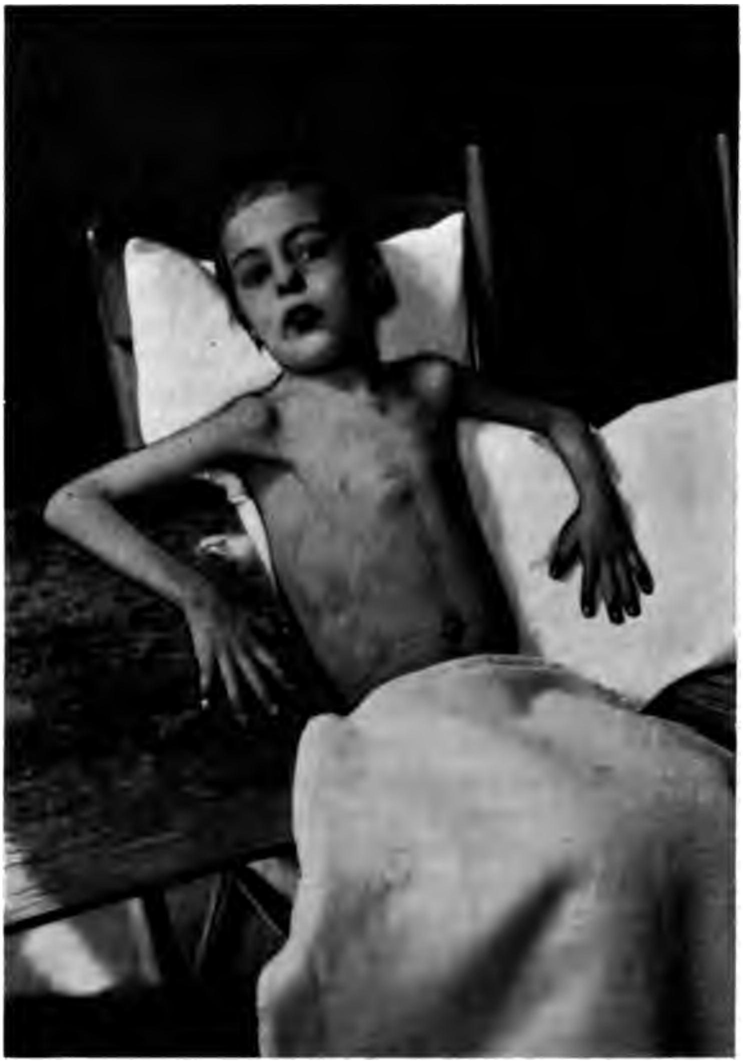


Fig. 106.—Shows the Cyanosis of Congenital Heart Disease, the Drum-Stick Finger-Tips, the Bulging Præcordia, and the Distention of the Abdomen in its Upper Zone due to Hepatic Congestion.

patients complain of symptoms. It is worthy of note in this connection, however, that my patient, whose case was narrated in the chapter on Pulmonary Stenosis, denied having suffered any inconvenience from his cardiac lesion, although this was pronounced, and indeed was not aware of its existence until informed of it by myself. Even up to the last his symptoms were chiefly attributable to the tuberculosis of the lungs, which was secondary to his valvular disease.

Children who are born with serious disorders of the heart evince notable backwardness of development, both mental as well as bodily. Their intellectual processes are sluggish, and they learn to talk at a later age than do normal children. In stature they are usually stunted, even dwarfish, and they are apt to present certain striking peculiarities in appearance. The nostrils and lips are thick and protruding, and the chest is more or less deformed in consequence of bulging of the præcordia. There is marked clubbing of the fingers and toes with incurvation of the nails, so that by German authors they are likened to drum-sticks (*Trommelschlaeger*).

The most characteristic feature, however, in persons with congenital cardiac affections is cyanosis. This *morbus cœruleus* of old authors is a general but not uniform blueness of the skin and mucous membranes, which is sometimes of so deep a hue as to be actually purple. It is most intense in those parts that are naturally red—the lips, nostrils, ears, cheeks, nails, elbows, and knees. It is always intensified by exertion and during the act of coughing.

The cyanosis and other visible circulatory effects of congenital cardiac disease is well exhibited in Fig. 106, which is the copy of a photograph taken of a nine-year-old girl whom, through the courtesy of Drs. Houston and Breid, I had the privilege of seeing at the Maurice Porter Hospital for Children. The little patient had been a *blue baby* from birth, and was brought to the hospital on account of attacks of præcordial pain during which she moaned continuously and displayed signs of great cardiac feebleness. I saw her in one of these attacks and noted the following: Uniform bluish hue of the surface, excepting the lips and ends of the fingers, which were of a deep purple tint; pronounced emaciation of the extremities, with exquisitely bulbous terminal phalanges; pronounced prominence of the cardiac area and distention of the hepatic region as far as the umbilicus; turgescence of the external jugulars; rapid and extremely thready pulse; epigastric pulsation, but no œdema.

Superficial cardiac dulness was greatly increased in all directions, and deep-seated dulness was of a quadrangular outline, reaching from the second costal cartilage to the seventh in the median line, and from 2 inches outside of right sternal margin nearly to the left anterior axillary line. Its great breadth at its upper part over the auricles was especially noticeable. The heart-sounds were very feeble, and over the body of the heart was a scarcely audible yet apparently systolic murmur. When, however, a hypodermic injection of $\frac{1}{24}$ of a grain of morphine, given to relieve the patient's distress, had stimulated the heart and enabled it to partially empty its overdistended chambers, and the little sufferer had grown quiet, the bruit came out loud and distinct. It was then found to have its maximum intensity in the third left interspace, close to the sternum, and to possess a very short presystolic portion.

From the great dilatation of the auricles, the position and character of the murmur and evident signs of impeded venous circulation, it was thought likely that this was a case of patent foramen ovale, or other septum defect, but whether or not with any other lesion could not be determined. The congenital nature of the defect was attested by the plus percentage of hæmoglobin, which was 115 per cent, and the number of red cells, which were in the neighbourhood of 7,000,000.

It is needless to remark that cyanosis is not limited only to congenital heart-lesions, since it is also present at times in acquired cardiac disease. In the latter cases, however, it is never so intense.

Many attempts have been made to explain the occurrence of cyanosis, but as yet none is generally accepted as quite satisfactory. It has been attributed to venous stasis and to deficient oxygenation of the blood, and apropos of this theory it is stated by Vierordt that Moritz found the CO_2 increase to between 45 and 46 per cent. Romberg thinks the cyanosis may be attributed to the abnormal admixture of arterial and venous blood. The intensity of its hue is due to the dilatation of the capillaries (Vierordt), which takes place to a far greater extent than can be the case in those diseases in which stasis develops more rapidly.

Striking as is the tint of the integument, there are certain other changes in the blood that are still more remarkable. Toeniessen first announced that in cyanosis, examination of the blood shows an increase in its specific gravity and its corpuscular ingredients. His observations have been abundantly confirmed by numerous investigators. The specific gravity in a boy of ten years was found by Banholzer to be 1071.8, while the hæmoglobin was 160, the red cells 9,447,000. The white corpuscles have been repeatedly ascertained to be as high as 16,000. In a case of congenital defect recently observed by me, hæmoglobin was 115; total red cells per centimetre 7,120,000; total white cells per centimetre 10,400.

No theory to explain this peculiarity of the blood is generally accepted. Malassez appears to have demonstrated that the blood of the superficial parts contains a greater number of red cells than does that of the deeper parts, and accordingly Penzoldt concludes this difference is due to an evaporation of fluids at the surface. This theory of a thickening of the blood, which Romberg mentions as having been established by Krehl, is objected to by Gibson, and I think justly, on the ground that the volume of the blood would have to be reduced at least a half in those cases in which the number of red corpuscles is doubled.

It has also been claimed that this augmentation in the number of coloured corpuscles is a compensatory process on the part of nature in order thereby to supply more oxygen to the tissues, and also provide a more adequate means of having the CO_2 removed. Gibson's hypothesis is so ingenious that it is here quoted at length. "In venous stasis the corpuscles are insufficiently oxygenated, they cannot thoroughly perform their duties as oxygen carriers, and they cannot yield so much oxygen to the tissues. It must further be remembered that in cyanosis there is less metabolism of the tissues, and therefore less waste produced. In a word, the functions of the corpuscles being lessened, the tear and wear which they undergo is reduced, and the duration of their individual existence increased. The number of the corpuscles must in this way be proportionately augmented, and this must lead to the numerical increase, as well as to the high percentage of hæmoglobin, until a balance is struck between the production and destruction of the blood-corpuscles."

In contrast to the usual results of blood examinations in these cases Mouillé is cited by Vierordt as having found a *reduction*, the red cells ranging between 3,500,000 and 4,500,000, yet this in no way invalidates the general proposition that the corpuscular elements are increased in cyanosis. Finally, it should be stated that a similar though less striking increase is observable in cyanosis in acquired heart disorders.

Laennec and Rokitansky attributed to cyanosis a protective influence against the development of pulmonary tuberculosis. Their views are erroneous, however, since it is a well-known fact, as has been stated in the chapter on Pulmonary Stenosis, that patients with this affection, in which cyanosis is particularly apt to occur, are especially prone to tuberculous disease of the lungs.

Another symptom in cases of cyanosis is coldness of the skin, particularly of the extremities, and hence these patients are remarkably sensitive to cool atmospheres. They are also very subject to dyspnœa and often manifest pronounced shortness of breath on comparatively trifling exertion, as was present in my case; but this, as we have seen, is a symptom common to all forms of cardiac disease in the stage of defective compensation. In these cases, when dyspnœa is a marked feature, there is usually evidence of considerable visceral stasis. In congenital cases, on the contrary, breathlessness is not infrequently pronounced out of all proportion to the signs of engorgement in the various organs, aside from the capillary dilatation emphasized by Vierordt.

This lack of such venous stasis as would ordinarily be expected in cardiac disorders of such evident gravity, is attributed by Romberg to the slowness with which the veins have been required to accommodate themselves to their abnormal burden (*ueberlastung*). Nevertheless, the deficient arterial blood-supply and the sluggish return of venous blood and the defective metabolism lead to disturbances of function on the part of the various viscera more or less severe and commensurate with heart-power. The variations in the pulse will be spoken of in connection with the physical signs now to be considered.

Physical Signs.—*Inspection.*—This is of special value only in the cases in which there are cyanosis, a dwarfish appearance, clubbing of the fingers, præcordial bulging, and other signs of a long-standing circulatory embarrassment. In such a case, moreover, there is usually the history that the patient "was a blue baby." Scrutiny of the cardiac area may detect displacement of the apex indicative of hypertrophy, but in all this there is nothing to attest the exact nature of the lesion. In not severe cases of congenital disease, as persistence of the ductus or patency of the foramen ovale, there may be nothing whatever in the patient's aspect to suggest the existence of cardiac mischief.

Palpation.—Of the serious congenital defects which come to a clinical recognition stenosis of the pulmonary orifice or conus is by far the most frequent, and it is in this affection that palpation is of special value. This usually detects a systolic thrill in the second and third left intercostal spaces close to the sternum. This may be so soft and weak as to be scarcely perceptible, or so coarse and strong as to tickle the hand. In patency of the foramen, of the duct, or even of the interventricular sæptum, there may be no thrill unless associated with some obstructive lesion, as just mentioned.

For the most part authors pay but little attention to the pulse, since it is thought to possess no distinctive characters. It should, however, be given particular study in cases of pulmonary stenosis, since, according to Starck and Renvers, its volume assists in determining the question whether or not there is closure of the interventricular sæptum. If the sæptum is perfect the supply of blood to the left heart is diminished, and hence the pulses of the upper extremities are small. When, on the contrary, communication exists between the ventricles, a side channel is provided by which the left ventricle receives a large supply of blood, and hence the pulses are of greater volume. Consequently, if in a given case of pulmonary constriction the pulse shows a degree of strength and volume out of proportion to what would be naturally expected, it suggests the likelihood of incomplete closure of one or both of the septa.

Kolisko is reported to have stated that when persistence of Botalli's duct exists secondary to atresia or great narrowing of the isthmus of the aorta or to congenital stenosis of its ostium, the pulses in the lower extremities are larger than those in the upper. This is due to the fact that the arteries given off from the aortic arch receive an abnormally small volume of blood, whereas a portion of the blood pent up in the pulmonary artery and intended for the ascending aorta through the left ventricle is switched off through the patent duct and enters the descending aorta, thus supplying the lower extremities with a disproportionate share of blood.

Percussion.—As in acquired heart-disease this means of investigation should not be neglected, since it is of extreme importance to discover possible modifications of cardiac dulness. In pulmonary obstruction the absolute and relative dulness are both increased to the right and downward in consequence of the right-ventricle hypertrophy. In patent foramen ovale and a defective ventricular sæptum the cardiac outline may or may not be increased transversely, according to the severity of the lesion. When the ventricular sæptum is incomplete the greater blood-pressure in the left ventricle forces a portion of the contents through into the cavity of the right ventricle. This chamber becomes surcharged, and tends therefore to hypertrophy and dilatation, which condition is shown by increase of cardiac dulness in that direction. Nevertheless, in both patency of the sæptum and foramen unassociated with other lesions præcordial dulness may in some cases remain normal.

Auscultation.—This usually furnishes the most valuable information concerning the presence and nature of these congenital affections by the detection of a murmur. Yet in cases of sæptum defects, including of course the foramen, there may be no murmur of any kind. When such a bruit exists, it is usually a loud systolic murmur heard throughout the cardiac area, particularly over the base. It does not appear to be limited to any area, as are the murmurs of acquired valvular disease; and this fact, when noted, possesses a certain amount of value.

Robert Maguire thinks that the systolic bruit of a defective ventricular sæptum is most distinct over the situation of the interventricular groove, and decreases in intensity as the stethoscope recedes from this line in either direction. As, however, the only case he has reported, so far as I have been able to learn, has not yet come to a necropsy, the proof of his contention is wanting, and although the statement may appear plausible, it cannot yet be accepted unreservedly.

Worcester has reported a case of patency of the foramen ovale, together with a small defect in the interventricular sæptum just below the right semilunar valves, which was discovered post mortem in a negro of fifty-seven who died of general paralysis. Several years before there was detected a long loud systolic murmur audible over the entire chest. The absence of symptoms during life is to be inferred from the fact that he served as a soldier during the civil war. The heart was found only moderately hypertrophied. There is nothing, therefore, distinctive of the murmur of foramen or ventricular sæptum patency. Cabot speaks of the quality of the bruit as harsh and vibrant; but there is in this statement nothing at all distinctive. In the case of a boy recently seen by me there was a loud systolic murmur not traceable to any particular ostium.

For a description of the murmur of pulmonary stenosis, as well as the other signs, the reader is referred to the chapter on that subject.

The auscultatory phenomena due to persistence of Botalli's duct are best described in the narration of a case I had under observation for several years, and which finally came to necropsy. The patient was an undersized woman of twenty-one who suffered from breathlessness upon rapid walking and an uncomfortable pounding of the heart. Her mother reported her as having been a small delicate baby, but as not having shown cyanosis even during fits of crying. Her only illness had been scarlatina at the age of nine. The radial pulses were small, regular, equal, and in rate between 90 and 100. There was no cyanosis or venous turgescence. The præcordium was prominent, particularly at the left of the sternum, but was not pigeon-breasted. The apex-beat was in the sixth left interspace, 2 inches from the sternum, strong and diffused.

There was a soft, not very distinct thrill in the second and third left interspaces close to the sternum, which was not synchronous with either systole or diastole, but was most pronounced at the end of expiration and beginning of inspiration. It seemed to follow the apex-shock by a very brief instant, and to run into the long pause. Absolute cardiac dulness was but slightly increased, whereas the relative appeared rather too broad. The heart-sounds were feeble and obscured by a loud harsh murmur that seemed to be systolic and audible throughout the entire præcordia, but most plainly at the base, and was transmitted to the lower angle of the left scapula.

Upon closer observation it was perceived that at the site of the thrill the murmur became a continuous remitting roar, having its maximum intensity just after the first sound and its minimum towards the end of the long silence, but never entirely ceasing.

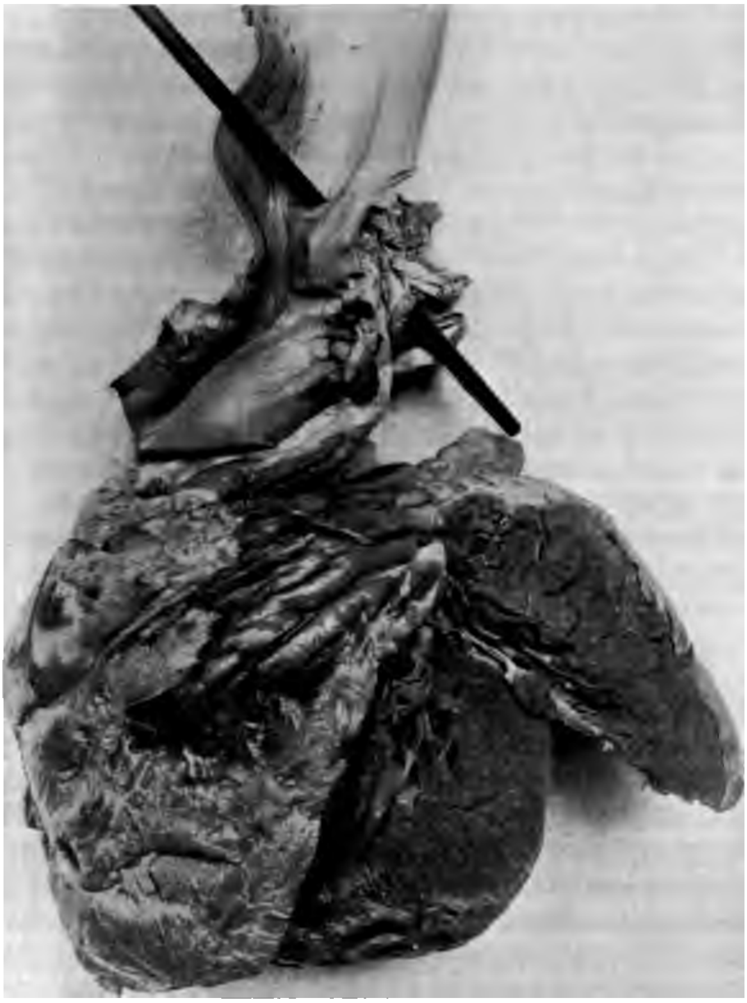


Fig. 107.—Heart from Case on p. 698, showing Concentric Hypertrophy of Left Ventricle and Sound passed through Patent Ductus Arteriosus.

Everywhere the quality of the bruit seemed to be the same. The lungs, abdomen, and urine were negative, but the blood examination showed a pronounced reduction in the percentage of hæmoglobin.

The precise nature of this lesion was not clear, but was evidently congenital. In time, however, the affection was decided to be either patency of the foramen or of the ductus arteriosus. As compensation appeared threatened, appropriate treatment was instituted, and soon a satisfactory degree of hypertrophy became reestablished.

To make a long story short, this patient ultimately married and was delivered of a child, passing through both pregnancy and labour without special difficulty. Unfortunately she became infected through the carelessness of her nurse, and died of septicæmia in the second week of her puerperium.

The necropsy was made by Dr. W. A. Evans, who found foci of suppuration in the right kidney and liver, but no evidence of inflammation in the cardiac structures. The specimen is presented in Fig. 107. The left ventricle was concentrically hypertrophied, its wall measuring 22 millimetres. The wall of the right ventricle measured 11 millimetres, and was therefore also thicker than normal. Both septa were complete and the foramen was not patent. All four sets of valves were healthy, but the aortic orifice was so small as to barely admit the index finger. This was found, however, to correspond in size to the lumen of the artery, which was abnormally narrow throughout. The circumference of the aortic ring was 43 millimetres; of the aorta, just central to branches, 45 millimetres; at opening of ductus, 43 millimetres; and 6 centimetres beyond, 40 millimetres. Of pulmonary ring, 55 millimetres; and of pulmonary artery, 55 millimetres.

The ductus was patulous, and upon searching for the cause of this persistence it was found that, instead of the isthmus being constricted, or the aortic arch smaller than the portion of the artery below the origin of the duct, it was as a matter of fact half a centimetre wider.

In this case the narrowing of the aorta below the origin of the duct, slight as it was, was yet sufficient to cause a portion of the blood-wave to be diverted into the duct and through it into the pulmonary artery, thus giving rise to the murmur and thrill. The left-ventricle hypertrophy was secondary to the aortic narrowing. This was an instance of chlorosis aortica, and accounted for the fact that treatment had never been able to restore the hæmoglobin to its normal percentage.

I have under observation at the present time two other patients, one a woman, the other a young man, who present almost identical physical signs and who, I believe, are also instances of this same congenital anomaly.

Diagnosis.—As there are several affections embraced by the term Congenital Cardiac Affections, it would be wearisome and unnecessary to recapitulate the physical signs by which each may be diagnosticated, and hence the reader is referred to what is stated above under the caption of *physical signs.* It only needs to be here stated that the congenital nature of the affection must be determined by the history and in some cases by a blood examination. If there is a history of the individual having been " a blue baby " or of his having been feeble from birth with evidence of circulatory embarrassment directly after birth, and if the child's appearance corresponds more or less to that described under *inspection,* there is strong likelihood of the cardiac disease being congenital. In many instances the parents are able to state that the family doctor discovered signs of heart-disease as soon as the infant was born or in its earliest weeks of life.

If the person presents well-marked cyanosis, and if examination of the blood discloses the changes previously described—i. e., an increase of hæmoglobin and red corpuscles over the normal—the diagnosis of a congenital defect can be positively made. In some cases, as of pulmonary stenosis, there may be nothing to prove conclusively during life whether the disease is congenital or acquired. In such a case, however, probabilities are always in favour of its prenatal origin, owing to the great rarity of the acquired form.

Finally, in doubtful cases of persistence of Botalli's duct, it is stated that by means of the fluoroscope a positive diagnosis may be made.

Prognosis.—As stated in the symptomatology of persistence of the foramen, this abnormality may occasion no signs of its presence, and patients may reach an advanced age, and die of some intercurrent affection. Unassociated with an affection of the pulmonic or other orifice, a defective sæptum ventriculorum or a patulous ductus arteriosus may also in no wise affect the prospect of longevity. It is far otherwise, however, as regards pulmonary stenosis. Even when the patient does not succumb to the heart-lesion directly, he is most likely to develop tuberculosis of the lungs. In comparing the gravity of this with other forms of congenital cardiac disease, excepting, of course, the uncomplicated sæptum anomalies just mentioned, Romberg states that up to the twelfth year of life affections of the pulmonary ostium and conus constitute three-fifths of all cases, whereas after the twelfth year, owing to the mortality of other lesions, these comprise four-fifths of the cases. Taking all forms of congenital cardiac defects together, he cites Stoelker's figures, which, condensed, are as follows: Out of 79 cases of all kinds, 24 died in the first six months of life, 42 had died before the end of the first year, 56 before the tenth year, and 71 had died before the twentieth year of life was reached.

It should be remembered, moreover, that, according to Kussmaul, congenital disorders of the heart predispose to endocarditis. In other respects the prognosis is influenced favourably or not by all those conditions of environment that have been fully considered in previous chapters. Lastly, when compensation has once begun to fail in these cases there is small prospect of much being accomplished by treatment.

Treatment.—As may be inferred from the preceding sentence, this must be largely or wholly symptomatic—that is, in accordance with the indications of each case. The reader is referred, therefore, to the discussion of the management of valvular diseases in general for the principles of treatment.

SECTION IV

CARDIAC NEUROSES

SYN.: FUNCTIONAL DISORDERS OF THE HEART

CHAPTER XXX

PALPITATION, TACHYCARDIA, CARDIAC PAIN, PSEUDO-ANGINA PECTORIS

Pathology.—There is a class of disorders which manifest themselves clinically by a perverted action of the heart, or by pain and other sensations in the cardiac region, or by a combination of the two, yet in which no structural alteration of the organ can be detected. They are often spoken of, therefore, as functional disorders of the heart. Objection is made to this term on the ground that in organic cardiac disease there is a disturbance of function, and, strictly speaking, such affections may also be designated functional derangements. Furthermore, it cannot be affirmed absolutely that some as yet undiscoverable alteration of the structure of the heart does not underlie or attend its perversion of function. However logical such reasoning may be, the term functional has been sanctioned by usage, and is generally understood by the profession and the laity to mean an affection which is not associated with demonstrable structural lesion. For this very reason it is often advisable in speaking to the patient or his friends to designate the disturbance as functional. A fear or an exaggerated notion of the gravity of the complaint may thus be allayed. Although from force of habit I frequently speak of these affections as functional, I yet prefer the designation cardiac neuroses, since one cannot observe these cases without coming to the conclusion that the manifestations on the side of the heart are the expression of a disorder of the nervous system.

One may be unable to detect any definite pathological lesion underlying this disturbance of the nervous mechanism, and yet it cannot be doubted that some neurosis is responsible for the cardiac symptoms. In some instances the disorder of the heart's action points to vagus influence, while in others the accelerator nerves of the heart are responsible for the manifestations. The exciting cause may or may not be discoverable, but an attentive study of the history and close analysis of the symptoms during and between attacks render no other conclusion tenable than that the cardiac and circulatory phenomena are secondary and subordinate to some disturbance of the nervous system, and hence outside the cardio-vascular apparatus.

It would no doubt be more in accord with the pathology of these cases to relegate these so-called cardiac neuroses to the domain of neurology, where they properly belong; but the symptoms calling attention to the heart are so often the dominant ones that they mislead the patient into the belief he has heart-disease. Indeed, the correct interpretation of the sensations is often puzzling to the physician, and hence it is customary to consider these cases in works of this kind.

Romberg classifies them as neurasthenic, hysterical, and reflex, in accordance with the nervous disorder underlying them. This would be well if all cases belonged strictly to these categories, or if the pathology of these neuroses was clearly understood. Such is not the case, and therefore I prefer to describe the various manifestations without attempting to divide them according to their apparent etiology into special groups.

Symptoms

Palpitation.—This is a transient derangement of cardiac action characterized by an increase in both the frequency and force of its contractions. Without warning, the heart suddenly begins to beat in a more or less disordered manner, and to give to the individual the sensation of a pounding or knocking against the ribs. Whatever may be the variations in rate and rhythm in individual cases, it is this subjective consciousness of the heart's action that constitutes the special characteristic of an attack of palpitation, and it appears to be this feature which alarms the patient. The heart may be rapid, 120, 130, or more, or it may remain below 100, but whatever its rate its action is violent. In the matter of rhythm also there are differences. Ordinarily the pulse is regular, but it may be irregular in frequency and force, and may be even intermittent. When this is the case the individual is likely to be thrown into a state of great alarm.

Each time the heart intermits, it is announced by a sensation of the organ suddenly falling or sinking in the chest; it is often described as a "sinking feeling." This is succeeded the next instant by a powerful throb, a sensation as if the heart gave a flop or jumped up into the throat, and with this very uncomfortable feeling the patient is apt to make a sudden exclamation or outcry, and perhaps quickly press the hand against the præcordia, as if trying to grasp the refractory organ. The heart may then quiet down, or it may race off as madly as before. It appears to me that in strictly neurotic persons without any discoverable organic mischief it is more common for the heart's action at these times to be rapid and regular (tachycardia).

During the attack of palpitation there is often a violent throbbing or pulsation in the arteries of the neck or in the abdominal aorta, or in both situations. The hand placed against the præcordium readily appreciates the energetic beating of the organ, and not infrequently the eye perceives a rapid rising and falling of the cardiac region. As it is so often expressed by the friends, "you can see the heart beat through the clothes." If the radial pulse is examined during such an attack or "spell with the heart," to quote the popular phrase, it may be found full and quick, or if the rate be extremely rapid, small and feeble. Vaso-motor changes are also very apt to accompany the seizure. The face flushes or pales, as the case may be, and the hands and feet are usually cold.

One of the most typical examples of palpitation was presented in a young man who consulted me only a few days ago. He was twenty-two and an athlete of superb physique, standing 6 feet 2½ inches, weighing 200 pounds, and with muscles of steel. He is an expert boxer, and can endure an arduous sparring-match without palpitation or shortness of breath. Two years ago he passed through an unusually severe typhoid fever, from which he made a good recovery with the single exception of sudden attacks of rapid, violent beating of the heart, that almost invariably came on shortly after a meal. They were accompanied and followed by a feeling of exhaustion, and were, naturally enough, very alarming to both the patient and his family. The attacks were of frequent occurrence, sometimes daily. I examined the young man at that time and was unable to discover any indication whatever of cardiac disease.

The history of a recent severe typhoid fever made me consider the possibility either of an acute myocarditis during his illness or of the myocardium having been seriously enfeebled in consequence of fatty degeneration, such as has been so well described by Quain. But the heart's dulness and the heart-sounds were normal, and inquiry elicited the statement that he was able to exercise, indeed had but just returned from a shooting trip in the mountains of North Carolina, without experiencing any shortness of breath, vertigo, or palpitation. The pulse was rapid during my examination, but its volume and force were excellent. I therefore assured him that his attacks were of a functional nature and did not indicate heart-disease.

His flesh at that time was rather too flabby, and he said he had been gaining weight rapidly since his recovery from his fever. Minute inquiry into his habits, diet, etc., brought out the fact that he was eating enormously and altogether too much carbohydrates, and was in the habit of drinking a large amount of water with his meals. He acknowledged some feeling of being bloated after eating. It was concluded, in the absence of other etiological factors, that gastronomic errors were at the bottom of his complaint, and he was advised to cut out his sweets and starches, to limit his consumption of fluids at meals, to drink lithia water between meals, and to begin his former systematic exercise both in the gymnasium and out of doors.

This *régime* was faithfully carried out, with the result that his palpitations almost entirely disappeared. During the following two years he came to see me twice, once a few weeks after his initial visit, merely to report progress, and the second time to receive an examination for life insurance, which on my recommendation was granted him.

This past week, however, he came again with his father, who said he wanted to know how it could be that so robust a young man could still have his attacks of palpitation without there being something wrong with his heart. He then explained that the Sunday previous his son was about to start for church with his mother, when all at once he was discovered by his father lying on the floor and his heart beating so fast and hard that it could be seen through the clothes. The attack lasted about twenty minutes.

The young man then spoke up and said he did not see any use of being concerned about the affair, as he knew perfectly well what had brought the attack on. He had eaten too hearty a breakfast, consisting of coffee and three pieces of German coffee cake, besides fried chicken and fruit. An examination was then made, and a more normal heart I have never listened to. The pulse was steady, regular, and 80, standing. The apex-beat was in the normal situation, absolute dulness was not increased, and the relative measured 3 inches to the left and 1 inch to the right of the sternum. The sounds were clear, of normal relative intensity, and entirely free from murmurs of any kind.

It was without hesitation, therefore, that the opinion expressed two years previously was reiterated. It was not quite clear why the attacks should take place in so powerful and an apparently perfectly well young man, but there was certainly an etiological connection between the attacks and indiscretions in the way of a too liberal allowance of carbohydrates. There was either a temporary abeyance of vagus control or a stimulation of the accelerator nerves of the heart. Whether this was an instance of reflex irritation or of some toxic influence resulting from indigestion, was not at all clear. But it would be ordinarily classified as a reflex cardiac neurosis.

In the foregoing case præcordial pain or other sensations of an allied nature were never complained of. It is quite common for an attack of palpitation to be accompanied by a painful sensation in the region of the heart or for the exaggerated cardiac action to follow the pain. At other times the patient may complain of the heart's pulsations as painful. In still other cases the chief complaint is of an indescribable feeling of distress or discomfort "at the heart," which is usually but not invariably attended or succeeded by palpitation. Such symptoms are frequent in individuals who are hysterical. This class of cases is well illustrated by the following example:

A physician, aged twenty-four, height 6 feet 1 inch, weight

156 pounds, gave a history of "heart-weakness" for a year. His parents, brothers, and sisters were all living and in good health and free from neurotic tendency, so far as the patient knew. With exception of measles in childhood he had never been ill, and he denied venereal disease or sexual excess, and did not use tobacco, alcohol, or narcotics.

During the summer of 1899 he had been particularly hard worked in his profession, and compelled to lose much sleep. In November he suddenly developed attacks of pain in the region of the heart that were speedily followed by accelerated forcible beating of the organ. It seemed to him that every throb of the heart produced pain just below the left nipple. These attacks were precipitated by exertion, such as walking, or even a long drive into the country. After they had endured for about ten days he became so bad that he used to faint away during his attacks, and he remained unconscious for an hour or more in spite of efforts to revive him.

This statement made me suspicious that the so-called syncope was not in reality a true fainting fit, and he was asked if he became absolutely insensible to his surroundings, or whether or not he knew in a dim way what was being done to him. He then replied that he believed he was vaguely conscious of his surroundings at those times.

These attacks recurred for about four months, and were finally cured by the taking of $\frac{1}{4}$ of a grain of codeine 4 times daily during three weeks. The drug then had to be discontinued because of the obstinate constipation it occasioned. During those four months he was much troubled by insomnia. Since April, 1900, his condition had improved somewhat, but at the date of his examination by me, October, 1900, he was still unable to endure exertion because of the palpitation it evoked.

The young man was a blond, evidently highly nervous and not strong, since he lolled on the lounge in my office, as though too weak to sit up. His hands were cold and moist, and his arm trembled while the pulse was being examined. This was full, tense, regular, and varied from 105 to 110. The apex-beat was in the fifth left interspace well inside the nipple, and the strong, rather broad shock was accompanied by a coarse thrill. Absolute and relative cardiac dulness were normal, the latter measuring 3 inches to the left of the sternal margin and 1 inch outside the right sternal border.

The first sound at the apex was partially obscured by a rough vibrant murmur of whizzing quality, which was loudest in the erect position, disappeared in the right lateral decubitus, and was scarcely audible when the patient lay on his left side. It was increased in intensity at the end of deep inspiration and grew almost inaudible at the close of expiration. The second pulmonic sound was not accentuated.

The liver was not palpable and its dulness did not pass below the inferior costal margin, right nipple-line. The abdomen was negative. The patient reported his urine as negative, containing neither albumin nor sugar. He was not conscious of indigestion, and the bowels were not constipated.

The diagnosis was made of a cardiac neurosis with an accidental murmur and palpitation.

The patient was advised to spend the winter in the South, where he could be in the open air, to take moderate, regular exercise, and endeavour to build up his nervous system, and to school himself to regard his malady as not organic. In the way of medication he was advised to take strychnine, give up the use of digitalis and allied heart tonics. Up to the present writing I have had no further report from this case.

This patient illustrated another feature of hysterical patients with disordered heart action. He declared he was always conscious of its pulsations, and could tell how it was beating without having to feel his pulse. To test him in this matter I took hold of the wrist and counted the pulse, and then told him to count aloud his heart-beats. In this he utterly failed, and I became convinced that his sensations were imaginary. This is not always the case, however, for in some instances the cardiac action is sufficiently exaggerated to be perceived by the patient. Sometimes, too, when the pulse-tension is high the individual can perceive pulsations in the extremities.

The powerful influence of the imagination and the readiness with which an attack of palpitation can be elicited by trivial causes are illustrated by the following case:

A law student, aged twenty-four, sought advice because of palpitations since the age of fourteen. Family history was negative, and the patient had not suffered from any acute disease that might have led to endocarditis or pericarditis. He thought his trouble with his heart dated from his study of physiology in school, when he observed that his pulse was too rapid.

At all events, from that time on he has been subject to frequent attacks of violent, rapid beating of the heart, and has been told repeatedly that he had heart-disease. He is greatly frightened by his attacks, which often come on without apparent cause or when fatigued by study, during unwonted exercise and excitement, or even too close application to his books. He is greatly troubled with flatulence, and this often sets the heart to palpitating. During an attack he is exhausted, alarmed, and notices particularly a violent beating in the stomach. Pollutions occur every two or three weeks, and are followed next day by extreme weariness, nervousness, and liability to his palpitations.

Examination showed him to be a tall, slender man with thin chest and broad intercostal spaces. The abdomen was thin, rather scaphoid when in the dorsal decubitus, and the abdominal aorta pulsated visibly. There was gurgling in the course of the transverse colon, but no dilatation of the stomach, and no demonstrable enteroptosis. The pulse was full, soft, rapid, and regular. The action of the heart was excited and abnormally forcible, and the cervical arteries pulsated strongly.

The apex-beat was in the normal position, cardiac dulness was not increased, and the sounds were clear, but too ringing. No murmurs could be detected. During the examination the patient became very nervous and exhibited a fine tremor, the hands being warm and moist.

There could be no doubt of the nature of his fancied heart-disease, and he was emphatically assured that his trouble was a neurosis and that he need apprehend no danger from his attacks. It was concluded also that in this case there was a reflex element, and that the cause of his palpitations lay in such an excitability of the cardiac accelerator nerves that they were sensitive to conditions which would be wholly inadequate to arouse them in a normal individual. The chronicity of the affection made prognosis rather unfavourable.

So much suffering was caused by the pollutions that the urethra was explored, resulting in the detection of nothing more than hyperæsthesia of its posterior portion. Local treatment was instituted by the specialist to whom the patient was referred, but with no appreciable effect on his attacks of palpitation.

The following extremely instructive case exemplifies the association of a veritable phobia with a distinct hysterical element and a reflex irritation, or at all events an imaginary reflex irritation:

The patient was a German-American, married, aged twenty-six, of medium height and weight. She sought medical aid because of "weakness, palpitation, and sinking spells." Her family history was negative, excepting that her mother and sisters were nervous. The patient was doubtful concerning her having had the ordinary diseases of childhood, but denied rheumatism or other illness of an acute or infectious nature. Said she had had stomach trouble and been nervous all her life, and at ten years of age had heart-trouble that came from her stomach and persisted about a year. From that attack she recovered without treatment, and she remained well with exception of nervousness until two or three years before marriage, at which time she had some nervous trouble that lasted a year and a half.

During her pregnancy she suffered much with her heart and stomach. The confinement was difficult, necessitating the administration of chloroform and delivery with forceps. She thought the chloroform weakened her heart very much, and she was not strong enough to nurse her baby. That was two and a half years ago, and ever since she has been a nervous wreck.

She has "heart attacks" from exercise, excitement, and after eating. These have been much worse the last four months in spite of treatment, and last week came as often as twice a day. Excitement, as from anger or domestic wrangles, which unfortunately are too frequent, at once give her a sinking spell, and she lies exhausted for hours, her heart beating very rapidly at such times, occasionally as much as 160 a minute, in this respect resembling paroxysmal tachycardia. At times the taking of some article of food or a remedy which disagrees with her stomach will instantly produce palpitation with extreme exhaustion. At these times she is alarmed, but is speedily quieted and her pulse slows down upon the arrival of a physician. She thinks she has been relieved a little by strophanthus, and has very strong notions regarding the effect on her of certain medicaments—e. g., dilute hydrochloric acid, which twice gave her a violent and prolonged attack of palpitation.

Her symptoms are always more likely to occur about ten days before menstruation, but when the menses have become established, her heart is more quiet and she feels better. Attacks are also very apt to follow looseness of the bowels, and conversely are not so easily called forth when she is constipated. Ever since the birth of her child she has been subject to the appearance on her extremities of "spots that look just like bruises, are dark red, gradually grow yellowish and fade away." Of late she has eaten only beef and wine, because anything else produced gas on the stomach and the attacks of palpitation and sinking spells.

She notices some shortness of breath on fast walking and ascending stairs; has appetite, and could eat if her stomach would let her, and after eating her stomach feels heavy. The bowel movements are irregular, but menses are regular, lasting two days. Her sleep is disturbed by palpitation, and she nearly always feels dizzy. During the rapid heart's action she notices pulsation in the carotids and in the stomach, and she has a feeling as if her lungs filled up with blood, her face, feet, and hands are cold, and she feels also as if she could not breathe.

The attacks persist from half an hour to several hours, and are not followed by a flow of copious pale urine. In a word, there is, excepting severe pain, scarcely a sensation connected with the heart of which this highly neurotic and imaginative woman does not complain. It is apparent that she is only too glad and ready to talk and dilate upon her symptoms. She is sure she is going to die in one of her attacks.

Physical examination showed pulse 106, small, regular, but not of noticeably low tension, and carotids throbbed slightly. Apex-beat was in fifth left interspace, 3½ inches from midsternal line, and accompanied by a slight thrill, which disappeared in the recumbent posture, although the apex-shock became more defined. Relative cardiac dulness reached from 1 inch to right of sternum to 3½ inches to the left of that bone. The first sound was accompanied by a faint, short, high-pitched systolic whiff, which was of limited transmission upward and to right and was slightly louder at end of inspiration—and both pulmonic and aortic second sounds seemed accentuated. In the standing position the abdomen bulged relatively too much below and was too flat above the umbilicus. The right kidney descended to a little below the costal arch, but could not be grasped. The liver could not be made out as having dropped downward. Gastric tympany reached 3 inches below and 1 inch too far to right of the umbilicus, and there was splashing. The abdominal aorta pulsated with abnormal force, but could not be distinctly palpated. The abdominal viscera evidently dragged somewhat upon their supports, and gastroptosis was undoubtedly present. The pelvic organs were negative. The lungs were negative, and there was no œdema about the ankles.

A week subsequently, after having been limited to two meals a day, and having enjoyed a week of immunity from her attacks, her heart's action was found slow but somewhat irregular in frequency. The apex-thrill previously noted was discovered to be a short but very distinct presystolic one, and the first sound was unmistakably thumping. Upon the patient assuming the recumbent posture the second sound, exactly at the seat of apex, was doubled and a low-pitched short murmur accompanied the first sound. I was therefore forced to conclude that this patient had mitral stenosis. Nevertheless, her symptoms were those of a cardiac neurosis rather than of an organic lesion.

She was sent to a well-known neurologist, who reported that, although distinct hysterical stigmata were not discoverable, he yet believed there was an hysterical element in the case. In addition, I could not rid myself of the belief that the condition of the stomach and bowels had much to do with the production of her attacks. At one time they would follow an indigestible meal or a relaxation of the bowels sufficient to merit the term of diarrhœa, at another some emotional disturbance, as a quarrel with her husband or an ungratified sexual desire—in short, a considerable variety of disturbing causes.

This case gave me endless trouble and perplexity, until at last, acting on a hint furnished by her statements concerning the etiological influence of diarrhœa, I prescribed a combination of astringents which kept her bowel somewhat constipated. She then became more and more free from her fearful attacks, and with increasing freedom from them regained a measure of confidence, so that at the present writing, 1901, I have not seen her for nearly two months.

August, 1902, heart's action being quiet, there was entire absence of cardiac murmurs, and the organ was to all appearances free from disease. The presystolic murmur and doubling of second sound above noted must therefore have been of accidental origin and in some way due to the disturbed cardiac rhythm.

A clergyman's widow, German, consulted me because she was sure something was wrong with her heart, and she feared she was suffering from the disease her husband died of. This was Graves's disease, the man having been frequently seen by me during his life and final illness. For two years after his death she remained in her usual health, but about a year ago she began to suffer from "spells with her heart," which were brought on by excitement, and sometimes, she thought, by the taking of food that did not agree with her stomach, for with the eructation of gas the palpitation began. At still other times the attacks came on without any apparent cause.

The action of the heart was likened to "rope jumping." She feels a "clutch at the heart," then her heart begins, and the next moment she is "entirely gone," the face being "deathly pale and the hands cold as ice." Nothing relieves her so quickly as a little brandy. Last week she had two attacks. She said she often noticed a gurgling in the left side of her abdomen, and this gave her much uneasiness. Her appetite was poor, and she was in the habit of drinking coffee four or five times a day. She was constipated, but her menses were regular. Her account of her complaint was not that of a neurasthenic, and there was nothing in her symptoms or appearance to suggest that she belonged to that class of sufferers. Neither was there any history of neurotic disturbances in her family.

She was thirty years of age, rather spare, and of medium height. There was no throbbing of the carotids, no tremor, no perspirations, no enlargement of the thyroid—in short, no indication of Graves's disease. The pulse was 90, equal, regular, and of fair tension. The apex-beat was in the normal situation, cardiac dulness was normal, and the heart-sounds were normal excepting, perhaps, that the first was rather too ringing. There were no murmurs. The lungs were negative also. Within the abdomen was the interesting finding that accounted for her gurgling. The abdominal walls were relaxed, depending baglike and bulging in the hypogastrium, while the epigastric region was too flat. An indistinct splashing was elicited, and gastric tympany extended well down into the pelvis, but not more than an inch to the right of the median line. It began at the level of the ninth instead of the seventh costal cartilage, and was too long vertically in proportion to its lateral dimensions. The kidneys and other viscera could not be made out as prolapsed. She looked anæmic.

Here, then, was an individual whose organs were apparently normal with exception of the stomach, which was prolapsed but not dilated. No other condition could be discovered to account for her palpitations, and accordingly she was told that the attacks were probably due to the gastric disorder, perhaps intensified by the undue use of coffee. She was emphatically assured that her attacks were not dangerous and was ordered to secure an abdominal supporter, and so adjust her clothing as to avoid the dragging of her skirts upon the abdominal parietes and pressure upon her stomach.

She was given tincture of nux vomica before and dilute hydrochloric acid in essence of pepsin after meals. For the attack of palpitation with pallor of face and coldness of the extremities she was given tablets of nitroglycerin. Coffee was forbidden, and instructions were given regarding a simple and nourishing diet. At present writing the symptoms still persist, but are less severe.

Tachycardia.—The physician is frequently called on to treat cases of habitually rapid heart's action, which are so annoying to the patient by reason of his subjective consciousness of the same that they may be said to be a persistent palpitation. In many instances this is the exaggerated cardiac action of Graves's disease, yet it is so prominent a symptom that it brings the patient to the doctor in the belief that the heart is the real seat of the trouble. As exophthalmic goître is a disorder of the nervous system instead of the heart, it will not receive special consideration in this work. There is another class of cases, however, which likewise present tachycardia and attacks of palpitation as their main symptoms, and which because sometimes associated with thyroid enlargement would seem to be incomplete forms of Graves's disease. The individuals are highly nervous, easily agitated, manifest more or less tremor, and in some instances have a warm unduly perspiring skin. They do not show exophthalmos, and if goître is not present, it is often exceedingly difficult to say whether they belong to the category of exophthalmic goître or not. Most, if not all, the cases I have seen have been in women, who, as a rule, are below the age of forty.

I have frequently discovered enteroptosis in these persons, and I am not able to escape the conviction that there is some intimate etiological connection between this condition and that of the nervous system. In some there has been evidence of moderate cardiac hypertrophy and in others, not.

This form of cardiac neurosis, as it may not inaptly be termed, was well illustrated by the case of a married woman of twenty-seven who came for treatment on account of symptoms that made her fear heart-disease. One sister had died of pulmonary tuberculosis at the age of twenty-four, and I may say, in passing, that in my cases I have been struck by the frequency with which a history of consumption in some near relative has been obtained. The patient had not been in her previously good health since her last confinement, two and a half years before. Her home was in a remote suburb in a lonely situation, and as night approached and her husband did not return she regularly grew nervous and apprehensive. She had lost weight, and for a considerable time had noticed that her heart beat too fast. Recently it had taken to giving a "flop," and every time this occurred it threw her into a state of alarm and agitation. Her neck had grown full, but she had given this no attention in comparison with the action of her heart. She was a hearty feeder, and all her functions appeared to be normal. Physical examination showed pronounced enlargement of the thyroid, which was firm and without thrill. There was no prominence of the eyeballs, but there was a fine tremor and the pulse was so rapid, in consequence of extreme nervous agitation, that no attempt was made to count it. Cardiac impulse was exaggerated, but the area of dulness was not demonstrably increased, and the sounds were clear, ringing, and free from murmurs. The lungs were negative and there was no enteroptosis. The case was regarded as one of incomplete exophthalmic goître, and a guarded prognosis was expressed. The patient was assured that she had no heart-disease, and I observed that this assurance at once favourably influenced the heart's action and nervousness.

In conclusion, it may be stated that under the prolonged use of iodine to the neck and internal medication addressed to relief of symptoms and improvement of digestion and general health, this patient ultimately made a complete recovery, the thyroid becoming of normal size and the tachycardia disappearing entirely. There was evidently a neurosis in this case, as shown by the powerful domination of her emotions over the action of her heart. Whether there was any direct connection between the thyroid enlargement and the tachycardia and palpitations I am not able to say, but her nervousness certainly disappeared *pari passu* with the decrease in the size of the gland. Nevertheless, her pulse-rate was invariably influenced by the state of her digestion and elimination.

I recall another case of rapid and pounding cardiac action in a female who presented fine tremor of the extremities, but no other signs of Graves's disease, and who was ultimately found to be pregnant at the time. As she was positive that her symptoms had first attracted her notice after the cessation of her menses, and the heart's action quieted down somewhat as the pregnancy advanced, I have not been able to determine what the connection, if any, was between the two conditions. This patient was unmistakably neurotic, as shown by both her family and personal history. Whether such cases are instances of incipient or incomplete Graves's disease or not, they are instances of cardiac neurosis so far as the action of the heart is concerned.

The foregoing cases present some of the symptomatology of heart-neuroses so graphically that it was thought best to introduce them before considering the etiology, although in so doing the general plan of this work is departed from. It is believed they will throw some light on the causation of some of the most common manifestations of functional cardiac disorders. As there is no demonstrable alteration in the structure of the refractory organ in typical cases, there is no morbid anatomy to be described. If a valvular lesion or dilatation of the heart is found in a person displaying the symptoms of neurotic disturbance of cardiac action, a combination which is not at all infrequent, the anatomicopathological changes on the part of the heart are not to be regarded as dependent upon the neurosis. There may be an etiological connection in so far as the organic heart-lesion may, through its influence over metabolism, aid in the development or the maintenance of the neurosis and may help to explain the ease with which the heart's action is perturbed. It may also be claimed that the palpitation and tachycardia induce the dilatation; but in my experience these cases do not display permanent cardiac enlargement to an extent that calls for treatment.

As previously stated, *the pathology of palpitation is obscure.* It is argued that it may be due to loss of vagus control, which allows the accelerator to gain the ascendency, or that there may be stimulation of this latter independent of an abeyance of the inhibitory apparatus. Again, it is not at all certain that there may not exist some histological change in the heart structures or nerve-centres which may account for the readiness with which the action of the heart becomes disturbed under conditions that are inoperable in the healthy individual. As Romberg says, arteriosclerosis sometimes develops in neurasthenics at an unusually early period of life; and who can say that there may not be some connection between this fact and the cardiac manifestations? These are matters of speculation purely, and in the present state of our knowledge we must content ourselves with speculation and theory.

Cardiac Pain.—This is another exceedingly frequent symptom in neurotic patients who suffer from fancied disease of the heart. It possesses no uniformity in intensity or character, being in one case sharp and darting, in another dull and continuous. Its one feature, common to all, is its location in the heart-region, usually in close proximity to the left nipple. It is sometimes intensified or even evoked by exercise—e. g., sweeping, which calls into use the muscles of that portion of the chest. In some instances it seems to be influenced, in part at least, by atmospheric conditions. Very frequently this pain is associated with a feeling of anxiety or oppression in the præcordia, which, because it occasions a vague feeling of apprehension, is by the Germans called *herz angst*, or anxiety of the heart.

This sensation may be wholly independent of any demonstrable change in the heart action, but is apt to be attended by palpitation, coldness of the hands and feet, and other indications of vaso-motor disturbance. As stated, it may accompany, but as a rule it seems to replace, actual pain. Whatever the exact characters of this præcordial feeling, it is very unlike, and is not to be confounded with the painful seizures which are designated angina pectoris, whether true or false. The differences are so marked that no mistake ought to be made, and yet it is possible for the feeling of cardiac anxiety to be mistaken for the constriction of the chest present in grave angina and the feeling of apprehension for the sense of impending death.

Pseudo-angina Pectoris.—From the standpoint of scientific accuracy this term may be and doubtless is objectionable, since there can be no such thing as a false chest pain. Nevertheless, this term is sanctioned by the usage of the best writers in this country and Europe. It includes those præcordial pains which closely resemble attacks of coronary angina, and are therefore spoken of by Osler as "allied states." The essential difference between them lies in the fact that pseudo-angina is independent of structural disease of the heart or its nutrient vessels, and that it is not likely to cause sudden death.

In true angina there is some condition within the heart which initiates the stimulus sent to the nerve-centres. In the pseudo form the starting-point of the painful attack is, according to Huchard, not the heart, but some peripheral or visceral nerve, most commonly one of the intercostals. The impulse thence passes to the medulla, and there, reaching the sensory centres, evokes a sensation of pain that radiates into the chest or down the arm with phenomena that point to a coincident stimulation of the vaso-motor and vagus centres. Often it is some painful point on the chest, generally one in the region of the left nipple, which acts as the starting-place for an attack. Whether such is the pathology of these cases or not, it certainly seems to me to afford a fairly satisfactory explanation of the essential difference between these two forms of angina.

Writers recognise three great varieties of this neurotic form: the reflex, the vaso-motor, and the toxic. Of these, angina reflectoria is the most common, although no one can observe these cases without coming to the conclusion that they are all very apt to blend indistinguishably with each other. Irritation within the abdominal organs is thought to be the most common starting-point of an attack of the reflex variety, and yet the vaso-motor form may likewise find its origin in some disturbance within the abdomen as well as in any part of the body. Huchard dwells much on the toxic angina and finds its causation in toxic agencies introduced into the system from without, such as tobacco.

An attack of pseudo-angina pectoris is agonizing, and because it usually begins in the cardiac area it excites a feeling of fear or apprehension that is closely allied to a sense of impending death. Ordinarily, however, the patient admits that this feeling is subordinate to that of pain. This latter radiates throughout the chest and into the left arm, which is apt to feel numb and cold. There is often a "clutching feeling at the heart," and the patient is apt to have a sensation as if she were "sinking away." At such times the pulse is said to become "very low," by which seems to be meant slow and weak. In cases of the vaso-motor type the face is pale and anxious, the extremities cold and clammy, and the pulse is small, usually slow, and often irregular or intermittent.

The sufferer from pseudo-angina is not compelled to assume an erect, motionless attitude, as in true angina, but lying on the bed or couch moves about restlessly and moans or cries aloud with pain. It is this feature of the attack on which reliance is chiefly placed in the determination of its real nature. Exceptionally, patients pass into a cataleptic state, apparently though not actually unconscious, rigid, and it may be cold, presenting in this state an appearance which is very alarming to the friends, who think it presages speedy death. The attacks usually come on suddenly and without warning, frequently at night, but in some instances there are prodromata, such as chilliness, restlessness, or vague nervous sensations. Some authors state that this neurotic form of angina displays a tendency to periodicity by recurring at the same hour on successive days.

The duration of the seizures is longer than that of true angina, lasting for one or more hours; their departure is apt to leave the patient weak and exhausted. They may abate gradually or suddenly or they may terminate in an attack of violent palpitation. Numbness and helplessness of the arm into which the pain radiates are not infrequent sequels of a paroxysm. Patients are naturally terrified, not only by the seizure, but also by the prospect of its return. Its frequency of occurrence is variable, but usually the intervals of freedom from pain are not long. Huchard observed cases in which as many as 200 or 300 attacks were experienced in the course of a single year.

In *pure angina reflectoria* without vaso-motor phenomena irritation originates in some distant part and the pain radiates thence into the cardiac area, from which it spreads along the intercostal nerves and even into the left arm. There is usually an associated feeling of anxiety and constriction, but the neuralgic element is the more pronounced. Huchard cites a case, not observed by himself, however, in which the paroxysms of pain originated in the cicatrix of a wound received many years before. This was situated at the bend of the elbow, and the attack of pain was precipitated by movements of the joint, by friction of the clothing, and even by gentle stroking of the scar. Squeezing of the middle finger was also capable of arousing a paroxysm, and this fact together with their cure by acupuncture led him to conclude there must have been an hysterical element in the case. He also quotes the instance of an officer who experienced an attack of pseudo-angina in consequence of painful irritation of his foot by one of his decorations, which had fallen into his boot, and there remained during the day. In such an individual there must be a highly neurotic tendency. Osler narrates a single case in which this form of angina followed attacks of vomiting, and therefore appeared due to gastro-intestinal irritation. Such attacks have also been known to result from exposure to cold.

In the *vaso-motor form* the exciting cause may likewise be exposure to cold or even the washing of the hands in very cold water. In this group there are phenomena of widespread vaso-motor spasm as well as pain, as might be expected.

The strictly *toxic form* is exceedingly uncommon and presents considerable diversity as regards the severity of the attacks and the prominence of certain features. Pain is often subordinate to a feeling of anxiety or præcordial oppression and there is disturbed cardiac rhythm in the way of retardation or acceleration, irregularity, and intermittence of the pulse. In tobacco angina there may be vertigo, pallor, a contracted pulse, tendency to syncope, præcordial anxiety, coldness of the hands and feet, and cold perspirations. According to Huchard, there may be other associated symptoms which are referable to nicotine intoxication, as dizziness, tinnitus aurium, dysphagia, and cephalalgia, a sense of suffocation or dyspnœa, general weakness, cerebral confusion, spinal tenderness, and disorders of vision. Although anginal attacks from tobacco may be incomplete in all their manifestations, they are none the less severe, and may be of great intensity.

Etiology.—Although the automatic action of the heart probably depends upon some quality inherent in the cardiac muscle-cells, and not upon the nerve filaments or ganglia situated in the heart-walls, still there can be no doubt of the powerful influence of mental and nervous states upon cardiac action. The class of disorders now considered is generally thought independent of structural disease of the heart, although persons with organic cardiac lesions may undoubtedly present some symptoms closely akin to those of the so-called cardiac neuroses.

The predisposing causes of the so-called functional or neurotic disturbances of cardiac action and of the various sensations referable to the heart are those disorders, neurasthenia, hysteria, etc., which for want of definite knowledge of their pathology are called neuroses. Psychoses, such as hypochondria, may also be attended by disturbance of cardiac action and other symptoms referable to the heart. Consequently in every case of cardiac neurosis the physician should endeavour to ascertain and expect to deal with some such underlying neurotic or psychical element.

Heredity, age, and sex have an undoubted etiologic influence over functional disorders now considered. Most of these patients present a clear family history of neuroses, and some of them have manifested unstable cardiac action from childhood. It is particularly in the female sex that the symptoms which have been described are encountered, and yet some of the most pronounced cases are seen in young men. Women appea especially prone to these symptoms during the child-bearing period and at the menopause. Their attacks of palpitation, heart-pain, or what not, are very apt to be evoked during the days immediately preceding menstruation. This is not because of any direct etiologic connection between the two, but simply because at this time, as at the climacteric, the nervous system is more than usually unstable. Whatever serves to lower nerve tone, or otherwise deteriorate the general health, predisposes to cardiac neuroses, and therefore masturbation, excessive venery, loss of sleep, sorrow, worry, too close confinement to mental pursuits, are all predisposing factors.

The influences which act as exciting causes are too numerous and various and often too obscure to warrant the attempt to enumerate them in detail. Patients are very apt to speak of having a "nervous shock," by which may be meant some sudden start or fright, an unexpected piece of bad news, and the like. In many instances the mere suggestion, whether subjective or made by another, that they have heart-disease suffices to excite an attack of palpitation. This is particularly the case with hysterical subjects, and I have known a word casually dropped, by being wrongly understood to apply to himself, to throw such a person into a violent fit of palpitation with coldness of the hands and a feeling of intense anxiety. On the other hand, a reassuring word will sometimes as promptly quiet the action of the heart.

There is often a close connection between the taking of food or a remedy and the onset of symptoms. This is sometimes doubtless the result of suggestion, at others of the formation of products of indigestion, and when this latter is the case it is difficult to say whether it is through a reflex or mechanical action or is the effect of the absorption of toxins. The symptoms not infrequently come on so quickly that there would hardly seem to be time for the formation and action of toxins. In neurasthenic individuals fatigue is undoubtedly an exciting factor. I have known a woman to take a short walk and immediately upon her return to be seized by a sinking spell with either rapid or slow and feeble pulse and coldness of the extremities, symptoms easily thought to indicate heart-failure, yet in reality due not at all to cardiac weakness. Ordinarily in cardiac neuroses an attack of palpitation is not produced by a reasonable amount of exercise. In fact, moderate exercise, as walking, is more likely to quiet down the heart. Nevertheless exceptions may occur, as was the case with one of my patients, a highly neurotic young man without demonstrable signs of organic disease.

Cases of pseudo-angina reflectoria have been shown in the description of symptoms to result from irritation of the abdominal viscera, from irritation of a peripheral nerve, and undoubtedly also from disturbances within the pelvic organs. It is my opinion that the same sort of influences may excite an attack of palpitation instead of pain. The same thing is true, I believe, as regards the impressions which are said to arouse an attack of pseudo-angina through the vaso-motor centres. Palpitation of this origin is not common, however, any more than is pure vaso-motor pseudo-angina. Instances in which an attack of pain is called forth by washing the hands in too cold water or by the impression made by a cold wind upon the intercostal nerves are certainly exceptional. Of the toxic agencies accredited with the production of pseudo-angina pectoris, disordered action of the heart, præcordial anxiety, oppression, etc., tobacco is by far the most frequent. This influence of the weed is not very common, and yet I have under observation at the present time a gentleman who assures me that he cannot smoke a single pipeful of mild tobacco without feeling his heart beat more rapidly and strongly than ordinary.

Diagnosis.—In deciding the question whether or not a patient's symptoms warrant their being classified as a cardiac neurosis, one should bear in mind that they are independent of structural alteration of the heart, and are in reality one of the manifestations of a disordered nervous system. Consequently one must first seek in the personal and family history and by a careful analysis of the symptoms for evidence of hysteria, neurasthenia, or of a highly neurotic temperament, conditions which have been shown to possess an etiologic influence over the phenomena that form a clinical picture of cardiac neurosis.

This being so far as possible settled, it is next necessary to determine the presence or absence of organic heart-disease. If such can be excluded, and the patient belongs to the age and sex in which neuroses are most prevalent, a correct diagnosis cannot for the most part be difficult. If, on the contrary, structural alteration of the heart is detected, or if the patient has arrived at the time of life when myocardial degeneration is likely, then one should be most cautious about expressing a positive opinion. It is very possible that he has to do with a case in which there is a blending of neurosis with structural cardiac disease. In all instances, even in the young, one must carefully study the nature of the symptoms, carefully discriminating those pointing to instability of the nervous system from such as indicate cardiac asthenia. One should therefore inquire minutely concerning the effect of exercise, for although one cannot assert positively that physical effort is without influence upon symptoms in cardiac neuroses, still such is ordinarily the case. This applies as well to anomalies of cardiac action as to the differential diagnosis of pseudo-angina.

Furthermore, without wishing to set it down as an infallible guide, I desire to give it as the result of years of observation that, if the patient is not subjectively aware of disorders of his cardiac rhythm, there is probably myocardial disease even if objective proof of the same cannot be had. The reverse does not obtain. The matter of dyspnœa requires close study. Patients with cardiac inadequacy from whatever cause experience shortness of breath upon exertion and not during repose except in an advanced stage. Neurotic individuals, on the contrary, unless markedly neurasthenic, are able to walk without breathlessness, whereas they are very apt to complain that they are unable to draw a long breath, or that they feel a "catch in their breath." They breathe superficially, and every now and then take an unusually deep inspiration, which is followed by a feeling of great relief.

If one is summoned hastily to administer relief to a patient in an attack of palpitation, a sinking spell, etc., a correct diagnosis is not always easy at first. Valuable information may be obtained, however, from inquiry into the history as regards previous attacks, mode of onset, etc., and from attention to the absence of signs of organic heart-disease and of secondary stasis. Furthermore, the patient generally displays nervous agitation, fright, etc.

In those cases of palpitation which manifest throbbing of the aorta either in the episternal notch or in the epigastrium, the differential diagnosis from aneurysm may be made by attention to the following points: (1) The history of attacks of palpitation and their association with symptoms of neurasthenia or hysteria; (2) the age and sex of the patient, who is generally young and more often a female; (3) the absence of pain, of signs and symptoms of pressure, of a localized tumour having an expansile pulsation and thrill; (4) the absence of an area of dulness upon the manubrium sterni or at either side, or at some point along the course of the abdominal aorta; (5) the failure to detect the auscultatory phenomena, of bruit and accentuation of the vascular sounds usually present in aneurysm; (6) the evidence derived from the sphygmograph and the X-ray.

In determining the significance and nature of pain in the cardiac region one should meet with but little difficulty if he remembers the following points: (1) The absence in neurotic cases of signs of structural cardiac disease; (2) the spontaneous origin of the pain, independent of exercise or of any other evidently exciting cause; (3) the presence of painful areas in the course of the fourth and fifth intercostal nerves, shown by Head to be symptomatic of both functional and organic disorders of the stomach. These hyperæsthetic zones are generally found on the left side as follows: (A) near the left nipple, upon the fifth rib, or in the interspace immediately above or below; (B) another upon the fourth costal cartilage or in the fourth interspace near the sternum; (C) at the lower end of the sternum or upon its appendix. There are frequently other painful points upon the back near the inferior angle of the scapula. The tender areas symptomatic of disorders of the thoracic organs are, according to the same author, located higher up, being in front on the sternum near the level of the third costal cartilage, and on the third rib, or near by, just within the vertical mamillary line. When the tender points first mentioned are discovered close inquiry will usually elicit symptoms of indigestion or the so-called auto-infection.

For the most part the correct diagnosis of the pseudo-anginas is difficult only in patients at or after middle age, and in them the question is likely to be rendered still more difficult by the discovery of cardiac hypertrophy or arterial thickening. In such an event a positive diagnosis must often be deferred until time throws further light on the case. In most cases, however, a correct diagnosis is possible by the discovery: (1) That the attacks, as previously mentioned, arise independent of, and are as a rule uninfluenced by, physical effort; (2) the sufferer is not compelled to seek the erect posture, but frequently prefers to lie down; (3) he does not present a picture of silent motionless agony, but moans or cries aloud and moves about restlessly; (4) the attack is of much longer duration, often lasting several hours; (5) it is often possible to discover signs of peripheral disease that may exert a reflex influence or to get a history of influences that are operable through the vaso-motor system or act as a toxin. As regards tobacco, however, it should be needless to suggest that in middle-aged men who are smokers coronary sclerosis is much more likely to be the cause than is their tobacco.

Prognosis.—This is practically that of the underlying neurosis. These cases are often of very long standing, and therefore present a correspondingly unfavourable prospect of cure. In the young, when the case is unmistakably one of neurosis, the assurance can unhesitatingly be given that death will not result from the attack of palpitation or pseudo-angina. When the diagnosis is doubtful, the patient may, for the moral effect, be told that his cardiac symptoms are functional, yet the friends should be warned of the possibly grave nature of the case. In strictly neurotic subjects the prognosis depends, moreover, upon the possibility of the removal of all those influences of environment which unfavourably affect the patient. In Graves's disease, or those allied states associated with enteroptosis, the prospect of obtaining immunity from their tachycardia and palpitations is very unpromising.

Treatment.—This must be directed not alone to the relief of the paroxysms of palpitation or pain, but also to the removal if possible of the underlying neurosis. It is not the province of this work to discuss the management of neurasthenia and hysteria, and therefore the reader is referred to works dealing with the subject. It need only be remarked here that the physician who would successfully treat cardiac neuroses must command the entire confidence and respect of his patient, and he must use the influence thus gained for their proper moral management. He must display no hesitation or vacillation in his suggestions and no irresolution in their enforcement.

Treatment of the Attack.—In most instances the medical attendant first sees the patient in one of his seizures, and is therefore called on to act energetically and promptly. Yet he should never be in such haste that he cannot first gain a tolerably correct notion of the nature of the disorder. He should never display alarm, and even if he thinks so, he should never tell the patient he is in danger of dying. On the contrary, he should endeavour to reassure the patient both by word and the calmness of his manner. Whether the attack is one of palpitation merely, or one of intense pain, the treatment is essentially the same, for there are usually associated symptoms of vaso-motor disturbance.

Palpitation.—I have never been able to see the wisdom of resorting to digitalis or remedies of similar action for the arrest of an attack of palpitation. These remedies are slow of action, the attack is in most instances short-lived, and before the digitalis takes effect the tumultuous heart-action subsides spontaneously, or because some other measure has met the indication. If there are pallor of the countenance, coldness of the extremities, and a small contracted pulse, a rapidly diffusible stimulant is indicated. The arterioles should be dilated so as to cause warmth and flushing of the surface, even though the pulse be rapid as well as small. To this end nitroglycerin is efficient and usually affords prompt relief. It is better to dissolve a tablet or to drop a minim of a 1-per-cent solution on the tongue, for its action is more prompt than when swallowed. Whisky, ammonia, camphor, or even hot ginger tea or hot peppermint water may be given, while heat should also be applied to the extremities and præcordium. If instead of being cold the surface of the body is warm and the face flushed, pulse full and bounding, then diffusible stimulants are contra-indicated. It is now better to apply ice to the præcordium and to give a full dose of one of the bromides, with possibly 2 or 3 drops of tincture of aconite root. This may be followed by a dose of digitalis or strophanthus. In most cases fear plays an important part in maintaining the attack, and consequently the very presence of the doctor, provided his manner is calm and reassuring, will do much to aid the action of remedies. If the seizure is unusually refractory and the patient's agitation does not subside after a sufficiently long trial of the line of treatment indicated above, then it may be well to inject $\frac{1}{8}$ of a grain of morphine for its calmative effect.

The Attack of Pain.—If the præcordial distress is not sufficient to merit the term of pseudo-angina, being plainly a pleurodynia with cardiac anxiety, it may yield to the application of a sinapism or of simple heat to the chest. If there are signs of vaso-motor spasm, or if the pulse is weak and perhaps slow and irregular or intermittent, a rapidly acting stimulant of the kind mentioned above should be given.

If the paroxysm is a pseudo-angina either one of two remedies is indicated: nitroglycerin where there is arterial spasm, and morphine subcutaneously where there is or is not such spasm. This latter not only allays pain and acts as an efficient cardiac stimulant, but it calms the patient and promotes subsequent sleep. Nevertheless it is well to bear in mind that there is always danger of these neurotic patients, who suffer from frequent attacks of pain, learning to depend upon the drug, and thus in time becoming victims of the morphine habit. The same objection applies to the use of alcoholic stimulants for the treatment of an attack of palpitation, sinking spells, etc., and therefore it is better to rely on other harmless but equally effective stimulants.

In conclusion, the physician should search for and endeavour to remove all those sources of visceral or peripheral irritation which serve to disturb the nervous system between attacks or may seem to act as exciting causes.

Enteroptosis, dilatation of the stomach, digestive indiscretions, or any other condition that may account for the cardiac symptoms are to be treated in accordance with the principles applicable to such cases and the special indications of each case. Great amelioration and sometimes entire relief of the distressing attacks of palpitation follow so simple a measure as the wearing of a properly fitted abdominal supporter in cases of ptosis of the stomach or other viscera. In addition, attention must be paid to the clothing, that too tight skirt-bands or corsets may not increase the dragging of the abdominal contents upon their supports. Properly given, massage is often of much benefit in these cases.

Finally, in all cases the exciting causes should be carefully sought out and the patient impressed with the necessity of avoiding all those influences which may precipitate an attack. He should be told that if he is to get better he is to aid in his cure by obeying instructions to the letter, since medicines alone are incapable of eradicating his disorder.

CHAPTER XXXI

ESSENTIAL PAROXYSMAL TACHYCARDIA

This is a highly interesting and very puzzling derangement of the heart's action which has received much attention from the medical profession since 1867, the date, according to Herringham, of the publication by Payne Cotton of the first recorded case. This disorder of cardiac rhythm consists in exceeding rapidity of action, and occurs in attacks of variable duration and frequency, during which the heart-beats number 160 or more to the minute. Medical men in the British Isles have always been keen observers, and here again, as in angina pectoris and bradycardia, have signalized their powers of observation. Cotton, Edmunds, Watson, and Bowles led the way, and in the next few years other observations were recorded by Nunnely, Cavafy, and Farquharson. On the Continent we find the names of Tuchzek, Gerhardt, Bouveret, Oettinger, Probsting, and many others. Gerhardt suggested the term Tachycardia in the year 1881, and in 1888 Bouveret suggested the appellation Essential Paroxysmal Tachycardia, to distinguish cases in which the paroxysms of excessively rapid action furnished the only clinical evidence of cardiac disease.

Acceleration of the heart's action to 140, 150, and even to 170 in the minute is sometimes observed in cases of valvular lesion, and may be assumed to depend in some way upon structural alteration of the walls due to the valve defect. But in those cases which Bouveret characterizes as essential tachycardia there is no clinical evidence of cardiac disease, and therefore the paroxysms cannot be considered symptomatic. This distinction is objected to, however, by Herringham, Gibson, and others, who prefer to call the condition simply paroxysmal tachycardia, since this applies broadly to all cases in which typical attacks of excessively rapid action occur.

Clifford Allbutt objects to the term paroxysmal, saying, " The interpretation is that tachycardia is a fairly uniform symptom group; and, as one of its eminent characters is its paroxysmal occurrence, the addition of this qualification to the name is superfluous."

Pathology.—There are no anatomical changes that can be definitely associated with essential paroxysmal tachycardia, and likewise there is no established pathological basis upon which an explanation of the phenomenon may rest. Prior to 1897 six post-mortem observations had been made in this class of cases, but they failed to disclose any constant or uniform lesion. In one, the wall of the left ventricle was in a state of pronounced fibrous degeneration, and in two the hearts were extensively fatty, while in three others no special changes were noted aside from dilatation. As these alterations have been found over and over again, indeed, are very common in hearts that have never manifested this peculiar disorder of action, it is plain that there is nothing in these post-mortem findings to explain the occurrence of paroxysmal tachycardia. Consequently various theories have been offered to account for the attacks.

Tuchzek suggested paralysis of the vagus, and Nothnagel, irritation of the sympathetic, sufficient to overcome the controlling influence of the pneumogastric. It has also been suggested that there may be a combined action of the two, vagus paresis and accelerator stimulation. Objections are urged against all of these theories. Tuchzek's theory has been widely accepted, and yet experiments on animals have failed to produce so extreme a rapidity of heart-action as is seen in these attacks, and Allbutt believes that in man abeyance of the inhibitory control of the vagus would not send the pulse up beyond 120. Likewise, stimulation of the cardiac accelerator nerve is said not to increase the pulse-rate beyond 150. Ascribing the rapid action to a combination of both necessitates the assumption of some cause which acts simultaneously on both nerves, and this, in Allbutt's emphatic words, " sins against the economy of causes."

Bouveret's suggestion that it is a bulbo-spinal neurosis, and Talamon's that it is of an epilpetic nature, are both not acceptable. Samuel West has urged that the attacks are due to alterations in the myocardium, to which Herringham would add changes in the nerve endings situated in the heart, a view that seems to appeal strongly to Gibson. Other suggestions, as a neuritis, are of still less importance. They are all mere surmises; and in the present state of our knowledge, without numerous and careful necropsies to throw light on the anatomical changes underlying this interesting symptom or disease, whichever it may be, we can only say with positiveness that we know nothing concerning its true nature.

Etiology.—This is likewise obscure. Most of the recorded cases have been in adults. Of the 53 cases collected by Herringham, the age was stated in 40, and of these there were 7 instances in children, 12 between the ages of twenty and thirty, and 13 in the following decade of life, the remainder being in persons past forty. Age, therefore, cannot be said to exert special predisposing influence. Both sexes are subject to attacks, and although 30 of Herringham's collected instances were in males, the preponderance of this sex is so slight that it scarcely warrants the conclusion that in sex alone resides any predisposing influence.

That in some cases there may be an hereditary element appears to be established by Oettinger's case, since there was history of the same sort of attacks in three preceding generations. In some of the reported cases there has been a history of previous disease —rheumatism, influenza, diphtheria, malaria, anæmia—that may possibly have been a predisposing factor, but a definite relationship of this kind has not been established.

In the way of possible *exciting causes* have been a blow on the chest, fright or other strong emotion, and a sudden physical effort. Attacks have also followed disturbances in the digestive tract.

Romberg states that paroxysmal tachycardia rests on a nervous basis, and may arise reflexly from disorder in any of the viscera or may result from some cause acting directly through the central nervous system, and is independent of any demonstrable cardiac disease. Thus it is plain that after all has been written on the subject of its pathogenesis we are no wiser than we were before.

Features of the Paroxysm.—Two conditions are essential if rapid action of the heart is to be considered an instance of essential paroxysmal tachycardia: (1) The *apparently healthy* heart must beat at least 160 times a minute. (2) The onset and termination of the attack must be so sudden and abrupt as to give it the character of a paroxysm. Although a pulse-rate of less than 160 is frequently observed in persons with some structural disease of the heart, still in essential tachycardia the number of cardiac contractions is often vastly in excess of this number, running as high as 200, and in a few instances even to 300 a minute. The pulse is small, thready, and often uncountable, because the extreme frequency of the waves and the emptiness of the vessel cause the pulse-waves to run together in an indistinguishable manner. To determine the heart-rate, therefore, one must count the heart-beats by auscultation instead of by palpating a peripheral artery.

The rhythm of the contractions is usually regular, but irregularity and inequality in their force are sometimes observed. The extreme rapidity of the cardiac systoles is at the expense of their strength and efficiency, blood-waves of normal volume are not discharged into the aorta, the arterial system becomes relatively empty, and the pulse is one of strikingly low pressure. This is illustrated by the appended tracing (Fig. 108) kindly furnished me by Dr. E. F. Wells from one of his cases.

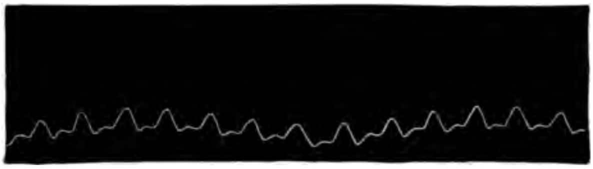


FIG. 108.—SPHYGMOGRAM FROM CASE OF PAROXYSMAL TACHYCARDIA.

The paroxysms begin abruptly and generally without premonition. Indeed, upon the occurrence of the first attack the patient does not always know what is the matter with him, and is only able to say he feels bad. If the tachycardia is short lived, the patient may experience nothing more than a vague feeling of discomfort and his outward appearance may not disclose anything unusual to the ordinary observer. There may be, however, pallor or flushing of the countenance. In some instances there are præcordial oppression and even pain, numbness or tingling of the arm (Gibson). Palpitation or fluttering of the heart may be complained of, and vertigo is sometimes experienced. Most sufferers from this complaint, notwithstanding repeated attacks and the fact that experience has shown the termination to be in sudden recovery, become greatly alarmed, and if the attack is prolonged to several days fall into a state of great mental and physical distress.

If the case is seen early there is usually so little evidence of the actual state of things in the patient's outward appearance that the medical attendant on feeling the radial pulse is usually struck with astonishment and even dismay at its rapidity.

If the attack lasts long enough it leads to cardiac inadequacy and the blood tends to accumulate in the heart-cavities. The heart becomes overdistended and the venous side of the circulation engorged, as shown by increased cardiac dulness, cyanosis, and it may be by pulsation of the jugulars (Gibson). There is pulmonary congestion, possibly also a small amount of œdema, and even albuminuria. The heart-sounds are feeble and the first at the apex may become almost inaudible.

In most cases the duration of the paroxysm is not sufficient to lead to such marked signs of stasis. The attack subsides suddenly after a few hours and the patient is left very much as he was before, feeling perhaps tired and dreading a recurrence, but able to return to his ordinary duties.

A striking peculiarity of such a paroxysm, whether long or short, is the persistence of the tachycardia even during sleep. Such attacks are usually repeated through a series of years, and yet cases have been observed in which but a single paroxysm was noted. Although recurrences are the rule, there is no regularity in their repetition.

Their duration is likewise variable, since the paroxysms may last from a few minutes to one or more days. In one or two instances the tachycardia persisted for two or even three weeks.

If the tachycardia is a symptom of some visceral disturbance, or, in other words, is a functional derangement of reflex origin, then it is easy to conceive of but a single attack and to understand how this may be of short duration. But, if it is due to some delicate and as yet unrecognisable alteration in the myocardium or bulb, then recurrences should be the rule, as indeed they are, and the attacks should be of considerable duration.

Diagnosis.—The determination of the fact of tachycardia is not difficult. The point to be decided is whether the rapid action is an instance of essential paroxysmal tachycardia or is of the kind called symptomatic. If it belongs to the former class, it should fulfil the following requirements: (1) A heart-rate for the time being of at least 160 a minute, (2) abruptness of onset and equal suddenness of termination, (3) failure to detect evidence of heart-disease either during or between attacks.

If, on the other hand, there is evidence of myocardial or endocardial disease, the tachycardia is symptomatic and not essential, no matter how rapid may be the pulse. In this class, however, it is not usual for the heart's action to exceed 150 a minute. Most instances of " heart hurry " belong to this class, and yet it is probable that the essential form occurs more often than is reported, either because the attacks come to the notice of the family doctor rather than of the consultant, or because the attacks are so transient that no physician is called in. Although I have repeatedly observed symptomatic tachycardia and have known several individuals who gave a history of the essential form, among them a medical man, I have not actually witnessed a paroxysm.

Prognosis.—In the essential form the prognosis may be said to be favourable so far as life is concerned. There is always an element of uncertainty in any case of extreme and protracted " heart hurry," but if a paroxysm terminates speedily no damage to the heart may be sustained. The real difficulty lies in the uncertainty of the length of time during which an attack may endure. In the aged, the feeble, and persons having a definite cardiac lesion such paroxysms are not devoid of danger. In most cases of paroxysmal tachycardia the seizure may be expected to terminate abruptly and spontaneously, but how long the patient is to remain immune from a repetition is a matter of too much uncertainty for the prudent physician to express an opinion. The history of cases shows that in most instances other attacks are to be expected.

Treatment.—The plain indication is if possible to arrest the paroxysm. This is called for, notwithstanding the fact that in the majority of cases the tachycardia has not caused death. Although a patient may have had repeated attacks that have ceased spontaneously, yet tachycardia is such an uncertain quantity that one can never be quite sure how another paroxysm may affect the heart. Unfortunately it is the same with this as with other maladies; our therapeutic resources do not always enable us to meet indications satisfactorily.

Theoretically, digitalis ought to enable us to slow down a runaway heart, but experience has shown its inefficiency in most cases. This remedy should not be administered recklessly in paroxysmal tachycardia, for if the attacks were to terminate spontaneously soon after the administration of a single very large dose or of several massive ones in quick succession, there might be positive danger of poisonous effects. If, as stated by Allbutt, digitalis produces diuresis even when it does not control the heart's action, it is likely to be eliminated and evil consequences will not result. Nevertheless, it is well not to administer more than 10 minims of the tincture hourly for six hours, and if at the end of this time no appreciable slowing of the pulse is produced, to have recourse to other means.

Ice may be applied to the præcordia, or one may try the effect of prolonged but not too vigorous friction of the skin over the upper portion of the spinal column, which has been said to slow the heart. The vagus may be compressed in the neck, or it may be stimulated by an electric current.

It has also been recommended that the patient take a deep inspiration, and then with his arms folded across the front of the chest and his feet pressed firmly against the foot-board of the bed to make a powerful expiratory effort while the glottis is kept closed. One of my patients who is a sufferer from essential paroxysmal tachycardia assures me that she has sometimes been able to check her heart by drawing a full breath, then while her body is flexed so as to compress her abdomen, making a powerful expiratory pressure. In her case also a paroxysm has been known to be arrested by the pouring of cold water over her wrists.

Whatever remedy is administered or whatever method of impressing the nervous system is tried, it is often found useless. It then becomes the physician's duty to support the heart until the tachycardia subsides spontaneously, and when cardiac dilatation sets in, this is imperative. To this end reliance must be placed on strychnine, caffeine, digitalis, etc., while the patient is kept at rest. The diet is to be simple and nourishing, and tea, coffee, or other stimulants are to be forbidden. In prolonged attacks it may be well also to administer a gentle cathartic.

It may not be possible to prevent recurrences, and attempts in that direction may seem to be something like firing in the dark, yet the patient should receive medical attention between attacks. In cases exhibiting subsequent signs of cardiac strain or in which there is an unstable nervous system, such regular treatment is specially advisable. Gibson recommends tonics, a course of the Nauheim baths with resistance gymnastics and such other measures as the experience of the medical attendant and the exigencies of each case suggests. In some instances it may be well to give digitalis or other heart-tonics for a long time. Every effort should be made to discover and remove any source of reflex irritation, and the daily life should be as healthful and free from excitement as possible.

SECTION V

DISEASES OF THE ARTERIAL SYSTEM

CHAPTER XXXII

ARTERIOSCLEROSIS

Degenerative changes in the coats of the blood-vessels were observed as long ago as the days of Senac and Morgagni, and by these investigators were described as an inflammatory process. It is to Rokitansky and Virchow, however, that we are indebted for thorough and systematic investigations concerning the origin and nature of the process to which Lobstein had previously given the name of arteriosclerosis. Virchow regarded it as a chronic arteritis and pointed out its similarity to the slow inflammatory process so often seen in the viscera, which is attended by the development of fibrous tissue. The inflammatory nature of arteriosclerosis was accepted by other pathologists also, but by certain of them was regarded as an evidence of some infectious process.

On the other hand, the cause of the sclerotic change was by Traube and others found in mechanical factors—i. e., in an increase of the arterial blood-pressure following persistent contraction of the arterioles. Indeed, some went so far as to attribute to high blood-pressure every case in which they recognised sclerosis and secondary cardiac hypertrophy.

The latest view of the pathology of this vascular change and the one that is coming into general acceptance is that of Thoma. Concisely stated, his conception of the process is that in consequence of lessened resistance of the media the vessel becomes widened with resulting slowing of the blood-stream. Connective tissue then develops in the subendothelial layers of the intima as a compensatory process by which to restore the normal relation between the artery and its contents. Although in most instances the vascular change is an attempt to make good a loss of elasticity and widening of the artery, still it may develop when the normal relation between the vessel and its contents is lost by reason of decrease in the volume of the blood. Romberg, to whom I am indebted for the historical data just given, finds Thoma's view highly satisfactory, since it seems to explain the development of arteriosclerosis in cases which were previously unaccountable by Traube's theory. Moreover, it has been founded on an immense amount of carefully studied material.

Morbid Anatomy.—Arteriosclerosis consists essentially in a degeneration of the media with secondary compensatory thickening of the intima. It may be localized, constituting the nodular form of Councilman, or it may be diffuse. In the nodular or circumscribed form whitish or yellowish patches are scattered along the inner surface of the vessel, which stand up from the surrounding level and are of a rounded contour. In the diffuse variety the arterial wall is stiff, and more or less dilated, while on the surface of the intima may be zones of nodular thickening and calcareous or atheromatous patches. In old persons the arteries are stiff, more or less tortuous and dilated. The inner surface presents numerous calcareous plates and atheromatous ulcers.

Examined microscopically, the thickening of the intima is found due to development of connective tissue between the endothelium and underlying elastic tissue. After a time, degenerative changes take place in this newly formed connective tissue which consist in hyaline transformation of the outer portion with areas nearer the endothelium of fine detritus in which fat droplets are seen. These areas of necrosis constitute the so-called atheromatous abscess. When these areas break into the lumen of the vessel depressions are left, known as atheromatous ulcers. The borders and bottoms of such ulcers are rough, and hence may become the seat of white thrombi. By the deposit of lime salts in these atheromatous patches calcareous plaques are formed which project above the surface of the intima, while by formation of chalky particles in the wall the artery may become transformed into a tube of almost bony hardness.

In the middle coat changes of a degenerative nature take place which lead to weakening and dilatation of the artery and consequent thickening of the intima. The middle tunic becomes thinned in consequence of atrophy and degeneration of its muscle-fibres and of more or less extensive destruction of its elastic elements. In some cases these elements disappear entirely and are replaced by connective tissue. The adventitia in its turn does not escape, but becomes infiltrated with round cells, especially in the neighbourhood of the *vasa vasorum*. The investing membrane becomes tough and fibrous and may also be of increased thickness.

The changes of arteriosclerosis which have been thus briefly described are not distributed uniformly in the affected vessel or in all parts of the arterial system. The lumen of small arteries is apt to be greatly narrowed and even obliterated by the hyperplasia of their coats, or it is blocked by thrombosis. The aorta and large arteries, on the contrary, are apt to become more or less dilated while their walls are rigid and the intima rough from the presence of calcareous plates and atheromatous patches, as previously described.

As already stated, the various parts of the arterial system are not equally involved in the sclerotic process. Thus Bregmann found as a result of analysis of the cases investigated under Thoma's direction, that the ulnar was involved in 94 per cent, anterior tibial in 93, subclavian in 88, cerebral arteries in 87, internal carotid in 87, radial in 86, splenic in 82, popliteal in 79, external carotid in 78, axillary in 71, femoral in 69, common carotid in 68, ascending aorta in 67, abdominal aorta in 64, external iliac in 58, and brachial in 55 per cent. This list shows some very remarkable differences which it is difficult to explain, and so far as I know have not been satisfactorily explained. Why, for instance, should there be so marked a discrepancy in the frequency with which the ulnar and radial are affected?

This matter will again be referred to in considering the etiology.

It should also be mentioned that arteriosclerosis of the nodular variety is encountered in some arteries with greater frequency than in others. These are such as do not run in straight or nearly straight directions, but make numerous turns in their course or give off branches at a sharp angle. The sclerotic process is here found at the points whence the branches depart or where the vessel undergoes a bend or curve. A glance at Bregmann's tables, quoted by Romberg, and compiled with special reference to the nodular form, shows that the abdominal aorta heads the list, while the common carotid, internal carotid, ascending aorta, and cerebral arteries follow close after in this order.

On the other hand, the radial is generally affected with the diffuse form, owing probably to its nearly direct course and the arrangement of its not numerous branches, conditions which permit uniformly high blood-pressure, and hence development of sclerosis throughout its length.

Associated with sclerotic changes in the vascular system are alterations of a similar nature in the various organs, particularly heart, kidneys, and liver. In the senile form the heart may be decreased in size, whereas in the diffuse variety, that encountered in comparatively young and robust men, the heart sometimes reaches enormous dimensions. Councilman found instances in which the heart weighed two and nearly three times the normal. The myocardium is apt to show fibrous degeneration, the coronaries to be sclerotic, and the aortic valve to be opaque, sclerotic, and in some cases incompetent.

The kidneys are especially likely to show the sclerotic change on microscopic examination, although to the naked eye the changes may be so slight as to be easily overlooked. The capsule is adherent and somewhat roughened on its surface, which may present dark red depressed areas due to atrophy. The capillaries of the glomeruli are thickened and may be obliterated and exhibit extensive hyaline degeneration. Atrophic changes may be present in the liver, particularly in connection with senile arteriosclerosis.

Etiology.—The great frequency of sclerotic changes in the arteries of old people very naturally attracted attention and suggested a close etiological connection between age and this disease. It has been thought directly due to senility, and hence a necessary part of advanced years. That arteriosclerosis is not an invariable accompaniment of age, however, is well known, and Gibson states that when Thomas Parr died at the age of one hundred and fifty-two his arteries were found by Harvey to be free from any evidence of degeneration. Such facts indicate that to the mere influence of age *per se* cannot be attributed the development of arterial degeneration. The explanation given by Romberg of the connection between the two conditions seems to me to be the best I have yet seen, and is, that when arteriosclerosis is found in an old man, it is because the conditions of blood-pressure which lead to the change have been operative during his many years, and therefore have come to manifest themselves more extensively than in a younger individual.

Males are without doubt more often and extensively affected with this change than are females. This is owing not to any special influence inherent in sex, but to the greater exposure of men to occupations, habits, and conditions of life in general which affect blood-pressure injuriously. The influence of occupations which necessitate arduous physical exertion, and thereby subject the arterial system to strain, has long been recognised and emphasized, particularly by the English. Thus day labourers, smiths, miners, etc., are very apt to develop arteriosclerosis, sometimes at a comparatively early age, and Romberg points out that in them it is the vessels of the extremities that are specially prone to disease. It is probable, also, that among the labouring classes other factors are at work beside physical toil, such as abuse of alcohol and syphilis. Nevertheless, strain of the vascular coats by severe and oft-repeated muscular effort cannot be ignored in the production of sclerosis.

Of diseases which lead to this degenerative process syphilis is perhaps the most important. Its relation to the form of endarteritis known as obliterans was described by Heubner, and is quite generally recognised. Chronic lead poisoning and chronic alcoholism are also recognised etiological factors, as is likewise gout. How these act is not quite clear, whether as suggested by Traube by causing persistent augmentation of blood-pressure or through the action of their poisons directly on the vascular coats. The excessive use of tobacco is also believed by some writers (Huchard, Romberg) to cause arteriosclerosis, particularly of the coronary arteries. Romberg likewise states that neurasthenic subjects are prone to arterial degeneration, as he believes, in consequence of the frequent alternations in blood-pressure occasioned by their unstable and excitable nervous state.

The manner in which these, and other predisposing conditions to be mentioned presently, act in the production of arterial degenerations has long been thought to be through the persistent increase of blood-pressure occasioned by them. Nevertheless explanation based on such hypothesis was not altogether satisfactory and did not clearly account for the pathology or etiology of the changes observed. In the light of Thoma's investigations and views, however, we are now able to understand much in the etiology which was before obscure.

It will be remembered that, according to his view, the thickening of the intima is an attempt at the preservation of the normal relation existing between the calibre of the vessel and the pressure of its contained blood. The loss of such proper relation or equilibrium, as it may be termed, is brought about either by dilatation of the artery in consequence of lessened elasticity or by diminution in the volume of the contents. Loss of elastic resistance on the part of the vessel is due to degeneration and atrophy of the elastic fibres of the media, and this destructive change in the middle coat may be due to the long continuance of excessive blood-pressure or to sudden, frequent alternations of blood-pressure.

Diminution of the volume of blood is seen very much less frequently, but is met with in the arteries of amputated extremities (Romberg), and, according to the same author, in the renal artery in interstitial nephritis. Of course the former requirement—i. e., increased pressure—is far more often and widely operative than is lessened blood-pressure. Accordingly, when we have to do clinically with arteriosclerosis we have to seek out some underlying condition, disease, occupation, or habit, that has caused long-continued and greater internal or endarterial strain than the vessel was able to bear. Slowly the middle coat has been forced to give way before the intravascular blood-pressure, *pari passu* the intima has taken on compensatory thickening and by degrees the sclerotic process has declared itself.

In some individuals blood-pressure has been abnormally high quite uniformly throughout the body and arteriosclerosis is general. More commonly, perhaps, the conditions influencing the change are local and the degeneration is confined to or at least far more pronounced in certain parts, as extremities, brain, coronaries, etc. For example, the frequency with which the anterior tibial is involved is explained by the fact that this artery is compelled to bear the distending weight of a column of blood which is heavy by reason of hydrostatic pressure (Romberg).

It has been frequently and forcibly pointed out (Fraenkel, Hasenfeld) that corpulent persons of a sedentary mode of life are especially prone to the development of sclerosis in the splenic, hepatic, and superior mesenteric arteries, and, according to Hasenfeld, earlier in these than elsewhere.

The explanation is, that owing to their sedentary pursuits and their habitual consumption of more food than the requirements of their inactive lives demand (luxus consumption) the vessels of their digestive organs are persistently overtaxed. In other words, blood-pressure within them is habitually too high. In time abnormally high and sustained pulse-tension is everywhere established, more or less wide-spread arteriosclerosis develops, and in consequence secondary cardiac hypertrophy (Fraentzel's idiopathic enlargement of the heart) results.

Another interesting phase of this question of blood-pressure relates to the development of sclerosis in vessels which are exposed to varying degrees of pressure, oscillations from low to high pressure, "*schwankungen*" (Romberg). Such alternations subject the artery to undue strain and probably account for the sclerotic change so frequently present in the arteries of the arms of workingmen. According to Romberg, they also explain the fact that sufferers from migraine sometimes manifest sclerosis of the arteries of the side of the head affected by the pain.

It is on this hypothesis likewise that we may explain the preponderance of arterisclerosis in the cerebral vessels of persons who are engaged in literary pursuits or whose occupations call for special activity on the part of the brain during a certain number of hours each day. May it not be for this reason that many an ambitious business man succumbs to the stress of modern commercial life? Romberg explains the greater frequency of coronary sclerosis in hypertrophied hearts as compared with those that are not hypertrophied, on the ground that coronary blood-pressure is higher in the former on account of their more forcible contractions.

If his view is correct, then one is tempted to query if the cardiac excitement experienced by stock-brokers and men of affairs under the influence of rapid fluctuations of the stock or grain market may not have much to do with the relatively great frequency of coronary angina in modern business men. In illustration of the important etiological influence exerted by variations of blood-pressure in circumscribed areas, Romberg cites the remarkable case reported by Erb of an ardent angler who developed a high degree of arteriosclerosis in the lower extremities, in consequence, it is thought, of his standing and walking for hours together in the cold water of the streams where he fished.

Additional instances of the injurious effect of long-continued high blood-pressure are seen in the degenerative changes found in the pulmonary artery of mitral patients and in chronic phthisis as well as the general arteriosclerosis of diabetic patients (Romberg). In short, upon the basis of Thoma's conclusions we are now able to understand many a case of arteriosclerosis the development of which was previously almost unintelligible.

Symptoms.—Arteriosclerosis is latent so long as it is of minor degree and not very wide-spread. When at length symptoms are produced, they depend upon the degree and distribution of the process and the organs affected. In some cases the clinical picture is that of renal inadequacy, in others of cardio-vascular disorder, in others again of disturbed cerebral circulation, and in still others of interference with the blood-supply to the extremities, digestive organs, or heart-muscle, as the case may be.

Sclerosis of the renal arteries may be secondary to already existing interstitial nephritis in consequence of diminished supply of blood to the renal capillaries, but in most cases it precedes or accompanies the development of the nephritis. The augmented blood-pressure occasioned, first declares itself clinically by increased secretion of urine, particularly at night. Examination of the urine in this early stage generally shows nothing more than a lowered specific gravity. When at length the sclerosis has become so extreme as to materially interfere with flow of blood in the renal capillaries, the urine grows scanty, and is apt to present characters like those of genuine contracting kidney.

In these cases there is apt to be more or less sclerosis of the arteries of other parts, particularly the heart, or general pulse tension becomes too high to be successfully combated by the hypertrophied left ventricle, and symptoms of cardiac incompetence are added to those of renal disease. Thus I recall the case of a middle-aged physician who, aside from cardiac breathlessness, developed symptoms of serious renal inadequacy. Urine grew persistently scanty, contained an occasional trace of albumin, but rarely casts.

He ultimately died with symptoms that were uræmic rather than cardiac, and the autopsy disclosed almost complete obliteration of the renal arteries.

In other cases the picture is that of slowly increasing, or perhaps suddenly induced, failure of heart-power, with renal symptoms of very inferior importance. Only to-day I examined a man of seventy-one who for two years past has noticed breathlessness, which of late has become serious cardiac dyspnœa. For more than twenty years he has had increased nocturnal micturition, but no other evidence of renal disease. He has been closely confined to his desk daily, and has been "a pretty heavy eater, particularly at breakfast." His chest is capacious and abdominal corpulence is quite marked. The radials, temporals, and carotids are stiff, and the heart is enormously enlarged, its impulse feeble, and its sounds distant and muffled. This case is a fair illustration of the etiology and symptomatology of the cases in which the clinical picture is what may be termed cardio-vascular, the chief, it may be the only, complaint being dyspnœa of effort.

Many such cases, like the foregoing, very well represent the clinical picture of chronic myocarditis. In others, symptoms of failing heart-power and of chronic interstitial nephritis are so intimately blended as to make it difficult to determine definitely which organ is the more seriously involved. In others again, glycosuria and renal cirrhosis precede the symptoms of vascular and cardiac disease, yet when the latter become marked they may dominate the scene. In all these cases, when cardiac incompetence supervenes, it is apt to prove most serious and to progress under the every-day appearance of increasing and unconquerable stasis, since the extreme degree of peripheral resistance incident to arterial rigidity renders restoration of heart-power impossible. They have been sufficiently portrayed in preceding pages and do not require repetition.

In comparatively few cases the symptoms are mainly, almost exclusively, referable to the arteriosclerosis as such. The arteries everywhere feel wiry and nodular, like a string of beads, or thickened and tortuous, and the pulse is small and weak. The superficial veins stand out prominently; the heart manifests slight if any change, being in some moderately and in others not at all enlarged; the urine is scanty and of poor quality, and if any albumin is present, it is a mere trace, while casts are scanty, being hyaline or granular; the patient complains of increasing inability to work or exercise; appetite and digestion fail; slight œdema appears at the ankles; the individual emaciates, grows sallow, pale, steadily more feeble, and at length takes to bed and dies from what appears to be general asthenia.

I have notes of such a typical case in an Englishman who was a farmer of about sixty-eight years of age. Up to a year or so prior to my seeing him he was hale and hearty, and able to perform active work of a not too severe kind.

Examination disclosed no distinct evidence of heart or renal disease, but the radials, ulnars, temporals, femorals, and tibials all felt hard and empty and most of them contained deposits of lime that gave them a pronounced beady character. Venous stasis was evident in the turgescent veins, palpable liver, and slight pitting of the ankles and shins. He did not complain especially of dyspnœa, but was much concerned over his growing weakness and loss of weight.

Treatment benefited him for a time, but he ultimately grew too feeble to report at my office, and as he resided in the country was lost sight of. It was ultimately learned, however, that he died after a few months of what appeared to be general feebleness with failing circulation. In his case, as in many, the heart seemed to be comparatively unaffected and the difficulty of circulation to be due to the impermeability, so to speak, of the arteries.

The rigidity of the arterial system interferes with proper discharge into the capillaries—neither are the arteries able to receive the full supply of blood sent from the veins, and stasis occurs.

In a considerable proportion of cases the clinical manifestations are not those of disturbed circulation in general, but of diminished or abolished blood-supply to a part, as the brain, extremities, heart, etc. The result is perverted function and structural alteration of a more or less serious kind. In some instances such disturbances are plainly apparent, while in others the manifestations of arterial degeneration are obscure and often misinterpreted or overlooked altogether.

Thus sclerosis of the cerebral arteries may be shown by impairment of memory and intellection, headache, transient vertigo, especially upon quickly assuming the erect position, change in disposition, increasing weakness, in a word, by the manifold symptoms due to cerebral anæmia or areas of softening (encephalomalacia) which result from the shutting off of blood-supply to definite areas. One should not forget also that when epilepsy develops at or after middle age, it may be due to arteriosclerosis within the brain (Hochhaus). Disease of these vessels is also a very frequent, according to Romberg the most frequent, cause of apoplexy. There may be either hæmorrhage into the brain from rupture of a miliary aneurysm, a condition of the arteries shown by Charcot to be very common, or the apoplectic seizure may result from thrombosis of a narrowed cerebral artery.

Sclerosis of the arteries in the medulla is a recognised cause of slowness of the pulse and of recurrent bradycardia known as Stokes-Adams disease, and which has been previously considered. (See page 627.)

Sclerosis of the arteries of the feet and legs is not uncommon, but apart from the change it creates in the elasticity of the vessel—i. e., stiffness and tortuosity, as perceived by the palpating finger—it does not often lead to serious disturbance of circulation in the region supplied by the sclerotic artery. The sclerosis may, however, according to Erb, be responsible for disorders of sensation and motion, vaso-motor and even trophic disorders. The first may be shown by paræsthesia, formication, pain, and a feeling of heat or coldness; disorders of motility, by intermittent lameness and extreme degrees of arterial narrowing by cramps, rigidity, etc.; vaso-motor disturbances, by coldness, pallor, cyanosis; and nutritional disorders, by circumscribed sloughing of the skin. In cases of obliteration from sclerosis, as is well known, there may be localized gangrene (senile gangrene).

According to Romberg, sensory and motor disturbances make their appearance at first only when the muscles are put in use—i. e., when there is a call for more blood to the part than can be furnished by the thickened arteries. In more advanced degenerations these disturbances are produced by insignificant movements, and at length the limb becomes stiff and useless.

Not only are vaso-motor neuroses, such as pain, redness, swelling, stiffness, etc., phenomena of arteriosclerosis, but, according to Romberg, there may appear symptoms of Reynaud's disease, cyanosis, pallor, and even gangrene of portions of the skin, such phenomena being particularly liable to affect the fingers. Fortunately, however, such serious disturbances are rare, and for the most part only minor degrees of sensory and vaso-motor perversions are present.

The heart may be affected by arteriosclerosis in either or both of two ways: It may be degenerated and feeble in consequence of thickening, narrowing or thrombosis of the coronaries, with angina pectoris and the symptom-complex of myocardial incompetence, or the heart may be secondarily hypertrophied in consequence of diffuse sclerosis of the arteries supplying the abdominal viscera. Hasenfeld has dwelt on the intimate connection between sclerosis of the mesenteric vessels and general cardiac hypertrophy, and Romberg also states that it is degeneration of these arteries which calls forth secondary hypertrophy of the left ventricle. Extensive vascular change of the brain and extremities may exist, he states, without appreciable enlargement of the heart. This coincides with my clinical experience, for the largest and most inadequate hearts I have ever seen have been in men whose abdominal corpulence and sedentary lives have furnished the conditions necessary for the development of vascular disease in the splanchnic area. Moreover, their stiffened radials and high-tension pulse have borne out the correctness of that assumption. On the other hand, I have seen old men with emaciated abdomens, peripheral arteries that were like wires strung with tiny beads, and feeble, even flickering pulses, and yet whose hearts could not be made out as hypertrophied. In some of these cases, to be sure, pulmonary emphysema renders the results of percussion uncertain, but the clinical picture is that of adynamia or of a cachexia, but not of myocardial failure, as in men of the other type.

Lastly, there is still another group of cases which present themselves in guise of chronic bronchitis and emphysema. They are usually at or past middle age, not confined to either sex, yet in my experience more often males of the labouring class. The vascular system is everywhere stiff, urine is of poor quality or may contain a small amount of albumin, and there is manifest hypertrophy of the right ventricle. This may be due in part to the emphysema, but a contributing factor of importance is the sclerosis of the pulmonary arteries. It is not always easy to determine whether this disease of the pulmonary vessels is primary or secondary, but as it is associated with retrograde change of the aortic system it is fair to assume that it plays a rôle in the causation of the emphysema and bronchial catarrh.

For the most part the course of this vascular disease is slow and indefinite. Years are usually consumed in its development, and even after symptoms appear the course is protracted or more or less rapid, according to the portion of the arterial system chiefly affected and to the degree of the sclerotic change.

Physical Signs.—*Inspection.*—There are two main types of individuals with arteriosclerosis. In one class they are large and imposing, more or less corpulent and with rather too flabby abdominal walls. In such, there may or may not be evidence of vascular disease in the peripheral arteries. The other type is quite the reverse. The individual is thin, looks ill-nourished, and the temporal, perhaps also the carotid, arteries are seen distinctly, the former looking like stiff tortuous cords and pulsating visibly. Superficial veins are also prominent, but cyanosis is not present. The only other information obtained by inspection relates to changes in the strength and location of the apex-beat, and to epigastric pulsation, signs which may be directly connected with arteriosclerosis, yet may be independent of the same.

Palpation.—This is the best and usually most reliable means of detecting arterial degeneration. If an artery which rests on a firm foundation, as the radial or tibial, is carefully palpated, it is perceived to be thicker and stiffer than normal. It can be rolled beneath the finger like a cord, and the vessel is difficult to compress. In many cases this is all, but in others the vessel is tortuous, and when the finger is passed along its course, presents small elevations that feel hard like beads, and hence lead us to speak of the vessel as beady. In some instances the artery shows minute elevations, which when carefully studied are found to be dilatations of the vascular wall—in other words, miliary aneurysms.

Particular attention should be paid to the cervical arteries, noting their position, size, regularity or smoothness, rigidity, etc., since changes in them may furnish valuable hints concerning the state of the aorta and inferentially of the coronaries. When the arch of the aorta is thin-walled and dilated it may sometimes be felt pulsating abnormally high up in the suprasternal fossa. Litten is authority for the statement that when the abdominal aorta is sclerotic and accessible to palpation, thrill is elicited by very much less pressure than is required if the vessel is healthy.

Percussion is of value only in the detection of changes in the size of the heart secondary to vascular disease. It may, therefore, by demonstrating hypertrophy of the left ventricle, afford a certain amount of corroborative information. Careful and deep percussion of the areas overlying the ascending portion of the aortic arch may detect a slight degree of dulness due to dilatation of the vessel. In such a case resonance is apt to be impaired in the first and second right interspaces close to the sternum. Dilatation and elongation of the arch may displace the heart downward, the same as does true aneurysm; and hence in cases in which the aorta is suspected of being sclerotic, it is well to percuss the heart carefully, with view, if possible, to ascertaining its exact location.

Auscultation.—Almost the only value of this means of investigation lies in the study of the second sound in the aortic area. This tone is normally more intense than is the pulmonic second in persons after thirty years of age, and hence it is the quality of this sound more than its mere intensification that is significant. General arteriosclerosis causes accentuation of the aortic second tone, but so also do other conditions, especially chronic interstitial nephritis. Taken in connection with left-ventricle hypertrophy, undue intensification of this sound is significant of arterial or renal disease or both. If the sound is not only intensified but is also sharply ringing, even of a metallic quality and is associated with stiff arteries in persons of middle age, it is generally considered to indicate sclerosis of the aorta. Should the sound be not quite pure, as well as accented, it is likely that the valve is also involved in the degenerative process. Not infrequently in persons whose vessels are resisting, there is a systolic murmur heard along the course of the ascending arch, and when present is, in the absence of signs and symptoms of aneurysm, to be regarded as due to roughening or dilatation or both of the aorta, not of stenosis of the ostium. Any other modifications of the cardiac sounds are indicative of secondary or associated changes in the heart-muscle and valves.

Diagnosis.—The recognition of sclerotic changes in peripheral vessels that can be reached by the palpating finger, as radial, ulnar, tibial, etc., is a very simple matter, and has been sufficiently described under palpation. It is far otherwise, however, with the diagnosis of sclerosis of the arteries within the cranial and other cavities. In such, diagnosis is usually a matter of inference instead of absolute demonstration, and must be arrived at by study of the patient's history, age, symptoms, etc. It is manifestly beyond the scope of this work to discuss the diagnosis of disease of the cerebral vessels. It may be stated, however, that tortuosity and rigidity of the temporals may, in connection with the head symptoms previously noted, be taken to point strongly to sclerosis of the cerebral arteries. If doubt still remains, or the external vessels are negative, the ophthalmoscope may be appealed to and is said to furnish early and reliable information concerning the state of the cerebral arteries (Thoma, Rehlmann, Koenig). The changes said to indicate sclerosis are pulsation and tortuosity of the retinal artery (when not due to chlorosis or anæmia), opacity of its coats, narrowing, and it may be thrombosis of the artery of the papilla, and miliary aneurysms and punctate hæmorrhages into the retina, the choroid, and the enveloping capsule of the optic nerve (Koenig).

The diagnosis of sclerosis of the aorta cannot always be definitely made. Romberg states that the condition of peripheral vessels, as radials, affords no criterion of that of the aorta, and hence stiffness of the arm or leg arteries does not warrant a conclusion that the aorta is also sclerotic. The state of the latter must be inferred, therefore, from careful study of the cervical vessels and of changes in the size of the heart or of its sounds. If the carotids appear healthy, if the heart is not appreciably enlarged nor displaced, and the aortic second tone is not unduly accentuated, then the aorta is probably healthy. If, on the contrary, the carotids are unyielding, the subclavians are situated abnormally high and feel stiff, if the left ventricle is hypertrophied, and lastly, but not least, if the aortic second sound is ringing and metallic, there is probably sclerosis of the ascending portion of the arch.

The question of the existence or not of arteriosclerosis within the domain of the splanchnic nerves may present great difficulties. The recognition of stiffened radials in a corpulent individual who complains of dyspnœa of effort out of proportion to recognisable changes in the heart, renders extremely probable a similar state of the vessels deeply situated in the abdominal cavity. If, on the contrary, accessible arteries are not stiff, one must depend for diagnosis on the history, symptoms, degree of blood-pressure, and adequacy as well as size of the heart. A history of sedentary pursuits and of luxus consumption; gradually developed and increasing shortness of breath; abdominal corpulence; high tension but slow pulse; cardiac hypertrophy without dilatation; these point strongly to arteriosclerosis as the cause of the symptoms.

In suspected cases one should test the efficiency of the heart-muscle as well as search carefully for indications of overstrain of the right ventricle. If the pulse is unduly rapid and feeble during repose, if cardiac dulness is increased transversely and downward with pulsation in the epigastrium, if superficial veins are engorged, there is reason to conclude that the heart is no longer quite adequate and that the dyspnœa is cardiac. Then if on the patient's making extra exertion, as by hopping about the room, the action of the heart grows unduly accelerated, perhaps irregular or intermittent, and the sounds become feeble, perhaps accompanied by an apex-murmur, but little doubt is to be entertained of myocardial insufficiency.

Even then the state of the internal vascular system may be a matter of doubt. Prolonged high tension of the pulse, as shown by Gaertner's tonometer, and a ringing metallic quality of the aortic second sound strengthen the assumption that the heart weakness is secondary to arteriosclerosis. It is, of course, presupposed that all other etiological data are wanting.

The differential diagnosis of such cases from the cardiac insufficiency of the obese (the so-called fatty heart), is often impossible, and, as a matter of fact, the two conditions are not infrequently combined. In the obese, however, there is a general distribution of adipose tissue far in excess of what exists where there is only excessive abdominal corpulence with arteriosclerosis.

Quite recently I examined a gentleman of fifty-three complaining of breathlessness on more than moderate exertion. His abdomen was very bulging and flabby, while his extremities and chest were rather thin, his radials were distinctly stiff, but the heart was not appreciably enlarged except on the left, and did not grow too rapid or irregular from the effort of hopping up and down my office. In this case I felt no hesitation in attributing his symptoms to arteriosclerosis, and not to myocardial inadequacy, particularly as his habits were such as tended inevitably to its development. Unfortunately all cases are not so clear, and hence necessitate great reserve.

With regard to the diagnosis of vascular disease in other inaccessible regions, it may be stated that coronary sclerosis is not often possible of positive recognition. It may be assumed when angina pectoris develops in a man past middle age, when there is reasonable evidence of sclerosis of the aorta and its great branches with subjective and objective symptoms of myocardial incompetence.

Sclerosis of the pulmonary artery cannot be diagnosed with any degree of certainty, but may be assumed if a patient with stiff arteries is a sufferer from chronic bronchitis and emphysema, and in addition there is unusual hypertrophy of the right ventricle.

Renal arteriosclerosis may be inferred if in conjunction with stiffened peripheral vessels there is nocturnal micturition, the urine being of poor quality. When in an advanced stage there is evidence of positive renal change, as shown by albumin and casts, it is practically impossible to say definitely how much is due to nephritis and how much to arteriosclerosis.

Prognosis.—This depends upon the degree of the vascular change discovered and upon the extent and nature of the visceral disturbance resulting therefrom. The process is inherently progressive, and I believe incurable. Symptoms may, however, sometimes be held in check by proper treatment.

Cardio-vascular symptoms are for the most part subject to the conditions which influence prognosis in cases of myocarditis, and need not here be dwelt upon. For the prognosis of cerebral and renal arteriosclerosis readers are referred to works devoted to diseases of the respective organs. The prognosis of sclerosis of the pulmonary vessels is essentially that of the cardiac or pulmonary affections to which it is secondary, while when vascular decay of the arteries of the extremities has once led to definite disturbance of circulation the prognosis is highly unfavourable. Progressive emaciation and loss of strength in general arteriosclerosis indicate so serious an interference with nutrition that it may be regarded as the commencement of the end.

Treatment.—This is to be divided into (1) prophylactic, (2) curative, and (3) symptomatic. The institution of *preventive* measures necessitate (A) the earliest possible recognition of vascular change and (B) the proper regulation of habits, diet, exercise, and excretion with a view to lessening undue vascular strain and correcting injurious fluctuations of blood-pressure. The disastrous effect of sedentary pursuits must be counteracted by appropriate gymnastic and abdominal exercises, including massage in cases of excessive abdominal corpulence. Heavy feeding must be restricted and its effects offset by outdoor exercise and sports, as golf, hunting, and fishing. Furthermore, the character of the dietary should be revised so as to exclude or reduce the eating of meats which are tissue-forming foods and are also harmful on account of the extractives they contain.

Theoretically, also, the diet should not consist of foods rich in lime-salts, and as a matter of fact Rumpf, of Hamburg, cuts out such articles from the dietary of his patients. He includes among such forbidden articles milk, eggs, cheese, rice, and spinach. For my part, I am of the opinion that quantity cuts a far greater figure than does quality, since it is a matter of every-day observation that the individuals who live the longest and are the most active with advancing years are those who eat sparingly and of a dietary relatively rich in vegetables, cereals, milk, and fruits. I regard it as a good indication when a person past middle age tends to lose weight gradually rather than to gain. Best of all, he should strive to hold his weight about at a standstill until well on in years.

Exercise in the open is a very important matter, especially for the man who having been accustomed to plenty of exercise in college suddenly finds himself tied down to his office desk many hours each day. He should endeavour in every way possible to get out for some sort of active physical exertion. If his profession or business duties tax his mental powers severely and keep him keyed up to the highest pitch day after day and month after month, then he should make whatever sacrifice is necessary to secure a yearly vacation, during which he can obtain perfect relaxation and recreation. Otherwise sclerosis of cerebral or other arteries will be his fate after middle age.

If, as is believed, the splanchnic nerves regulate blood-pressure and irritation of these nerves increases blood-pressure, particularly within the abdominal cavity, then, from a prophylactic standpoint, digestive derangements, including of course chronic constipation, should be corrected. This is desirable from another point of view —namely, that by improving excretion the system may be rid of toxines which may be of influence in augmenting arterial tension, and eventually leading to arteriosclerosis.

In a word, the prevention of degenerative changes in the blood-vessels calls for the removal, or at least the minimizing, of all injurious influences which are believed to derange blood-pressure, and thereby subject the vascular system locally and generally to strain.

The *curative* treatment of arteriosclerosis is, I believe, unpromising. The French, and of late some German clinicians, as Vierordt, express faith in the ability of iodine to arrest and even cure vascular degeneration. The remedy is administered in the form of iodide of sodium rather than of potassium because of its being better tolerated. It is begun in small doses, 2 or 3 grains twice or thrice a day at first, and as the system learns to tolerate the remedy it is gradually increased until 15 grains three times a day are reached. In this dose the iodide is continued over a long time—i. e., from eighteen months to three years—but with occasional intervals during which the drug is not taken. Vierordt is said to omit the remedy one week out of five and one month out of every five months. Given in this manner, and from the start increased so cautiously as not to cause undue irritation, he has seen very gratifying results. I have made repeated attempts to get my private patients to persevere in the use of iodide of sodium after the manner recommended by Vierordt, but always without success. It has invariably disordered appetite and digestion, and at length has had to be discontinued.

This therapeutic agent may favourably affect the patient's general condition, and even the vascular disease in cases of syphilitic origin (although I believe the claim is made that favourable results are obtained even when there is no specific taint), but it is very difficult to see how any drug can promote resolution of the sclerotic process, or why it should be well to do so. If the development of connective tissue in the intima is, as Thoma believes, a compensatory process by which is attempted to make good atrophic changes in the media, then how can any line of therapy be beneficial that does not restore the media to its former normal state? The iodide may in some way prevent or remove deposits of lime and the hyaline degeneration that ultimately occur in the newly formed connective tissue, but can it do more or would it be well to have it do more?

It seems to me, therefore, that when arteriosclerosis has once become pronounced, our efforts must be limited to the prevention or lessening of symptoms—in short, must be *symptomatic*. To do this we must endeavour to promote better circulation in the arterial system, since it is in this respect that evils arise.

Cardio-vascular derangements are to be combated in the same manner as disorders due primarily to cardiac insufficiency, and these do not need to be repeated. I should like to urge the necessity, however, of freely using vaso-dilators, as the nitrites, that if possible the arterial paths may be somewhat opened up and the labour of the left ventricle thereby lessened. In this class of cases nitroglycerin, etc., should always be given whenever it is necessary to resort to digitalis. It is because of the constricting action of digitalis on the arterioles that strophanthus ought to be tried instead, and only replaced by digitalis when it has proved inefficient.

Nothing is of greater service in cases of diffuse arteriosclerosis with secondary venous engorgement than a periodic purge by means of calomel. The catharsis should be brisk to be of benefit, for relief does not follow until several watery stools have been secured. I have seen truly surprising results from such simple treatment. One instance in particular comes to mind as I write, that of an old German with very stiff vessels who exhausted both my patience and my resources in a vain endeavour to procure relief from formication and coldness of the thighs. At last, in despair, I prescribed 5 grains each of calomel and jalap for the purpose of preventing his return to my clinic. He did not reappear for two or three months, when one day he returned, and on entering the clinic room exclaimed that he had come back for another powder, as he had never had anything do him so much good.

I recall also an Irishman with stiff arteries and an obstinate chronic bronchitis that had defied the efforts of several well-known practitioners, and who obtained greater relief from his dyspnœa and cough by a single dose of 5 grains of calomel than from all the cough mixtures he had previously taken.

Fraenkel reports the highly interesting and instructive case of a man with general arteriosclerosis who was relieved of his nocturnal asthma for a period of three months by a single sharp attack of epistaxis. This suggests that in cases of cardiac asthma, which so often form a distressing feature in the clinical history of arteriosclerosis of the cardio-vascular type, it might be well to resort to venesection when catharsis has failed of ameliorating the symptom.

In cerebral arteriosclerosis I am of the opinion that stimulants and vaso-dilators are indicated. Judgment and caution should be exercised in their administration, however, lest the heart be too vigorously stimulated and rupture of a miliary aneurysm result. The safety of such medication may be estimated by the state of the cardiac muscle. They are certainly indicated when the systoles are feeble and the brain is not sufficiently flushed.

The treatment of coronary sclerosis and its resulting angina pectoris has already been considered in the chapters on Chronic Myocarditis and Angina Pectoris.

The management of renal sclerosis is essentially that of chronic nephritis, since the two conditions are so frequently combined. When vascular disease in the extremities has led to gangrene, the treatment is of necessity surgical. Sclerosis of the pulmonary artery is practically that of arteriosclerosis in general plus that of bronchitis and emphysema.

Only general principles can here be laid down. In every case special symptoms must be left to the judgment of the practitioner.

CHAPTER XXXIII

ACUTE AORTITIS — ACUTE ARTERITIS — SYPHILITIC ARTERITIS—ENDARTERITIS OBLITERANS—PERIARTERITIS NODOSA—STENOSIS OF THE AORTA AND PULMONARY ARTERY—CONGENITAL SMALLNESS OF THE ARTERIES

I. ACUTE AORTITIS

When acute inflammation of the aorta is discovered it is in most cases associated with the changes of sclerosis in the same situation or with an acute endocarditis. French authors, however, have described a form of acute aortitis which they claim is independent of antecedent sclerotic change and occurs in the course of acute infectious diseases. Such statements are received with considerable reserve by the Germans, and von Schroetter in Nothnagel's System seems quite sceptical on the subject, particularly as regards its clinical recognition.

Morbid Anatomy.—The aorta is found more or less dilated, and the surface of the intima looks rough from the presence of reddish or grayish translucent more or less thickly scattered patches of a gelatinous consistency. These are minute thrombi, and it is through the detachment of these that cutaneous and other infarcts occur, the same as in acute valvulitis. Indeed, acute aortitis is so similar to acute endocarditis that the description of the latter may answer for the former.

The process may be of a benign type and proceed to the formation of so-called vegetations, or it may behave like ulcerative endocarditis and lead to serious destruction of the vessel and rupture. In this manner communication may be established between the aorta and one of the auricles, a contiguous vessel or the pericardium with fatal hæmorrhage (Romberg). The media and even the adventitia becomes infiltrated with round cells, and if the process is sufficiently prolonged newly formed vessels may penetrate into the intima.

As already stated, degenerative changes are usually found associated with the evidences of acute inflammation. There may also be an associated valvulitis affecting previously healthy valves or more often as an acute process ingrafted on an old-standing aortic-valve lesion. In other distant parts of the body there may be discovered local changes due to benign or septic emboli cast off from the aortic intima.

Etiology.—The aorta may become acutely inflamed in consequence of direct extension of an identical process of the endocardium. French clinicians (Huchard, Leger, Siredi, etc.) maintain that acute aortitis may arise in the course of scarlatina, measles, variola, independently of involvement of the endocardium, and Fiessinger is said to have seen it in a case of influenza (Gibson). The latter says also that acute aortitis may be associated with acute pneumonia, pleurisy, and pericarditis. The disease has also been attributed to trauma, and has been observed in the course of chronic nephritis.

Symptoms.—Acute aortitis in most instances is latent or is overlooked by reason of its occurrence in the course of some other distinctive affection. The case discovered by Thoma, and which occurred during measles, had produced no symptoms whatever. Von Schroetter appears to think that the clinical features described by Huchard in such a brilliant and interesting fashion are not to be attributed to acute aortitis *per se*, but are such as are so often observed in cases of arteriosclerosis affecting the aortic arch. In Chapter IV, page 158, I have depicted a case which I took to be acute endocarditis because of the subjective symptoms and clinical findings, and in which post-mortem examination disclosed an aortitis together with endocarditis, the acute process having developed on top of old sclerotic changes that had masqueraded under the guise of aortic insufficiency.

I will briefly portray the features that are claimed by the French to have been observed in acute aortitis unconnected with other aortic or endocardial lesions. Fever is usually absent, but if present it is due to the primary infection, not to the aortitis as such. The countenance is apt to be pale and anxious. The pulse is small and weak, regular or not, as circumstances in each case may dictate. The patient is likely to complain of pain in the upper sternal region, and sometimes extending through the mediastinum and down the back along the spinal column. The pain is described as burning, sticking, smarting, etc., and in some instances is said to radiate into the left shoulder and down the arm, very like that of angina pectoris. The resemblance to this latter is enhanced by a feeling of oppression and anxiety in some cases.

Peter has observed tenderness on pressure in the intercostal spaces to the left of the manubrium, and undue throbbing of the right subclavian has been noted by French writers (Laboulbene, Faure), and by them is attributed to the greater liability to inflammation of the left subclavian than of the innominate artery. Von Schroetter, however, believes that if such difference in the pulsation of the two subclavia exists, it is due to sclerotic changes, an opinion in which Gibson concurs.

Dysphagia, cough with expectoration, and disturbance of the digestive tract shown by vomiting and flatulent distention of the bowels, have been observed in some cases.

In short, the symptoms of this affection are often wholly wanting, and when present are not at all distinctive. There is nothing in them which may not be observed in other affections involving the heart, and hence Romberg states that even when the malignant form of acute aortitis occurs, there is nothing in its clinical picture to distinguish it from ulcerative endocarditis.

The course of acute aortitis is often protracted and the termination is usually in death.

Physical Signs are usually indefinite, or are such as are found in other acute inflammations involving the cardiac structures, or are those of the infection in the course of which acute aortitis occurs.

Inspection.—The countenance may be pallid and anxious, the carotids throb strongly, and the right subclavian may pulsate more powerfully than does the left.

Palpation is negative unless pressure elicits sensitiveness in the intercostal spaces to left of the sternum and along the course of the aorta.

Percussion is likely to be negative unless dulness be revealed at right of the manubrium in cases in which the inflammation leads to dilatation of the aortic arch.

Auscultation.—This is not likely to furnish information of a positive kind. A systolic murmur over the situation of the ascending arch may be evoked by dilatation of the vessel, and in cases in which the valve is also affected there may be impurity of the aortic second tone.

Diagnosis.—This can rarely if ever be more than conjectural. If the character of the pain and oppression simulate that of angina pectoris, it may possibly be differentiated from it by the fact that in acute aortitis this symptom is likely to persist, or at the most show only remissions, not intermissions.

The differentiation from acute endocarditis is not possible in all, perhaps not in most cases. Aid may be obtained, however, if one notes that in the course of a disease resembling endocarditis no changes in the area of cardiac dulness or in the heart-sounds are developed, or if on repeated examinations one should be able to detect increasing dulness over the ascending aorta indicative of dilatation. In my case this was noticed, but was not correctly interpreted, owing perhaps to the coincident dilatation of the right auricle.

Prognosis.—This may be said to be very unfavourable. The occurrence of embolic phenomena renders the outlook most unpromising. Rupture of the aorta is a possibility that should always be borne in mind in suspected cases of the disease.

Treatment cannot be expected to do more than relieve symptoms. Rest in bed is imperatively indicated, and the strength of the patient must be sustained by highly nourishing, easily digested food. Pain, when severe, should be allayed by morphine, counter-irritation, hot applications, etc. Nitroglycerin may be of service by diminishing intra-aortic blood-pressure, and strychnine is a valuable general and cardiac tonic. Digitalis is only useful in case of threatening cardiac inadequacy.

II. ACUTE ARTERITIS

Morbid Anatomy. — Circumscribed inflammation of the larger arteries is sometimes observed in connection with an inflammatory process of surrounding tissues or in consequence of embolic plugging. Infiltration with small round cells takes place in the outer and middle coats, later on also in the intima. The endothelial lining becomes swollen and of increased thickness, while the underlying layers of the intima show the development of newly formed connective tissue.

In cases in which the inflammation is the result of plugging, thrombosis also occurs, and in time the thrombus undergoes organization. If the embolus is infective, the inflammation may spread to the parts outside of the vessel and set up abscess. When the arteritis results from surrounding inflammation, thrombosis and subsequent organization may likewise take place.

The **etiology** has already been stated in the opening sentence. Acute arteritis results either from adjacent inflammation or from embolic occlusion.

Symptoms are likely to be recognised only when the arteritis is situated in an extremity or a part accessible to palpation, and when thereby one can detect either embolism or thrombosis, or when there is phlegmonous inflammation of the tissues surrounding an artery of considerable size.

When local inflammation invades the artery, involvement of the latter is likely to be masked by the symptoms of associated phlebitis. In the latter event there are circulatory disturbances due to interference with return flow, swelling, and more or less œdema, together with pain and great tenderness.

In the case of embolism there are pain and phenomena of obstructed circulation, coldness (local syncope), cyanosis, and numbness.

Physical Signs consist of such phenomena of local inflammation or of the accompanying phlebitis.

Inspection perceives swelling and redness of the affected extremity.

Palpation is of greater service. The limb is hot, painful to touch, usually pits on pressure, and at some point careful palpation is generally able to detect resistance due to the embolus or to thrombosis extending for a variable distance above the seat of the plug.

Diagnosis.—This is to be made by the history, local symptoms, and the result of palpation. The differentiation of acute arteritis from phlebitis is not always easy or possible.

Prognosis depends upon the nature of the cause and the completeness of collateral circulation. Acute meningitis is a possibility in certain cases, and of course affords a very grave outlook.

The prognosis is always unfavourable in cases in which the arteritis is secondary to acute malignant endocarditis.

The **treatment** of acute arteritis is partly medical and partly surgical. The affected limb should be elevated, kept at absolute rest, and enveloped in moist heat, as poultices to which anodyne remedies may have been added. Pain is to be allayed by local sedatives or by the use of opium in some form. Should an abscess occur, it is to receive appropriate surgical management.

III. SYPHILITIC ARTERITIS

Vascular changes observed in syphilitic subjects have been the object of careful study by numerous investigators, among whom should be mentioned Lancereaux, Heubner, Weigert, Doehle, Baumgarten, Vendeler. Some of the changes are unquestionably of luetic origin, while others are by some authors, as von Schroetter, accepted with considerable doubt.

Morbid Anatomy.—The inflammatory changes in the arteries are of a chronic nature and invade circumscribed portions of a vessel or are limited to the arteries of certain regions, as of the brain. The process may show itself as circumscribed patches of a grayish white translucent appearance, or the entire vessel may be changed into a whitish or grayish cord in consequence of the transformation of its coats into fibrous tissue. In this form the adventitia, and ultimately the media and intima, become infiltrated with round or fusiform cells. The process may remain in this stage of inflammatory infiltration (von Schroetter), but as a rule it goes on to formation of fibrous tissue in the several coats.

This hyperplasia of the walls is often extreme and leads to very considerable narrowing and even occlusion of the lumen of the artery.

In this respect syphilitic arteritis differs from arteriosclerosis, which is more apt to lead to dilatation than to obliteration of a vessel, although it may do this latter in the smallest arteries.

It has been shown, furthermore, particularly by Baumgarten, that minute gummata are scattered in the middle coat in immediate proximity to the vasa vasorum. Atrophy and rupture of the media result, and in time the rents are repaired by the formation of cicatricial tissue. The subsequent contraction of these areas leads to pouchings of the intima, which, when they are found in the ascending aorta, are almost pathognostic of syphilis (Romberg). These pouchings of the aortic intima may prove the starting-place of future aneurysms.

In another form of arterial disease due to syphilis the vessel becomes invaded by a syphilitic process in its neighbourhood. The vessel is surrounded by a gummatous mass or by dense cicatricial tissue, and the coats of the artery are more or less thickened and altered (Ziegler). In the early or inflammatory stage the outer and inner coats are rich in cells, but as the process advances fibrous tissue replaces the cells wholly or in part. The media is not so much invaded by fibrous tissue as are the adventitia and intima.

The cerebral arteries appear to be the ones most frequently affected. The aorta and coronary arteries may, however, be the seat of syphilitic disease, and in a few cases the vessels of the extremities have been affected. C. O. Weber is said by von Schroetter to have found the right branch of the pulmonary artery in a syphilitic girl greatly narrowed by reason of a gumma in its wall. Zeissl is also stated by the same author to have found the left brachial artery invaded by a gummatous infiltration, while Langenbeck saw the same sort of process in the right brachial of another case.

Etiology.—Syphilitic arteritis is a late manifestation of lues.

The **symptoms** are determined by the seat of the arteritis. In the case of the brain they are those of disturbed or obstructed circulation, loss of memory, dizziness, headache, mental confusion, epilepsy, etc.—in short, such as arise from areas of acute softening.

When the disease affects the aorta it may lead to aneurysm or to the symptom-complex of sclerosis of the arch.

Arteritis of this origin may be a cause of angina pectoris by leading to sclerosis and occlusion of the coronaries, particularly the left anterior descending branch. In very rare instances a coronary artery has been said to be invaded and obliterated by a gumma of the myocardium.

In the extremities syphilitic arteritis occasions clinical manifestations of obstructed circulation the same as may other forms of arterial disease, pallor or cyanosis, coldness, and eventually gangrene.

The **diagnosis** must depend upon the history of luetic infection and on the discovery of unmistakable lesions indicating a late stage of the disease. Even in such a case one cannot always say positively that the vascular changes observed are of specific origin. They may be due to arteriosclerosis and be independent of syphilis *per se*. In some cases one may be obliged to await the result of treatment before being able to arrive at a definite diagnosis.

The **prognosis** is not always favourable as regards recovery, although appropriate therapy may in some cases affect a restoration of health. If the arteritis has led to pronounced fibrous thickening and considerable obstruction, to pouching, or even to aneurysm, there is small prospect of favourably influencing the process by antisyphilitic medication no matter how vigorous.

The **treatment** should consist of the administration of approved specific remedies—i. e., mercury and iodides. In addition, one may have to treat certain symptoms, as angina pectoris, cardiac inadequacy, gangrene, cerebral disorders, etc. The management of aortic aneurysm will be found in a succeeding chapter.

IV. ENDARTERITIS OBLITERANS

The following account is a condensed statement taken from von Schroetter's excellent description of the disease in Nothnagel's Specielle Pathologie und Therapie. No apology for such a transcript is necessary, since the disease in question is rare, and comparatively few contributions to the subject have been made. The designation obliterans was suggested by Winiwarter, whose case is considered so typical by von Schroetter that he makes use of Winiwarter's description. Billroth gave it the name Hyperplastica, while Orth called it Productiva. Other observers to whose views or cases von Schroetter refers are Weiss, Brochard, Schlesinger, Sternberg, Wiedermann, Ortmann, Hadden, Goldflam, Weber, Collet, Chatin, Roque, Braun.

Morbid Anatomy.—The disease occurs most often in the smaller arteries of the foot or leg, occasionally also in the upper extremity, and exceptionally in other parts. Upon macroscopic inspection the vessels are seen to be enveloped by a tough fibrous sheath which binds them firmly together. The individual artery —e. g., posterior tibial—is converted into a firm whitish cord, and on section is seen to be filled with a whitish gray or grayish brown mass, so that a probe can be passed into the vessel only with difficulty or not at all.

The artery is nevertheless not uniformly so filled, yet on the whole is transformed into a rigid cord in consequence of its interior being filled with a somewhat yielding wide-meshed tissue. The process begins at the periphery and extends upward, reaching from the plantar peroneal and posterior tibial arteries, even in some instances to the femoral, or in the case of the arm, to the brachial.

Histological examination reveals in different places a somewhat variable condition, yet which is in reality a hyperplasia of the intima which may augment its thickness to even eight times the normal. In the larger vessels the newly formed connective tissue is composed of round, spindle-shaped, or stellate cells, between which can be recognised an intercellular substance made up of delicate threads. According to Winiwarter and others, several strata of elastic fibres may be seen in the outer portion of the intima next to the media. Finally, minute blood-vessels are seen to exist within the connective tissue of the interior, which Winiwarter regards as an extension or formation of new channels by which an attempt is made to provide a collateral circulation, and not as an organization of a thrombus. The capillaries thus formed permit a partial injection of the mass filling up the lumen of the artery. In spite of this attempt at a collateral circulation the stump after an amputation does not bleed freely when the Esmarch bandage is removed.

Etiology.—This is practically unknown. It has been observed in men far more frequently than in women, and what is especially strange about it is that it attacks comparatively young and previously healthy individuals. The process does not necessarily invade all the arteries of a limb, for it has been found in the posterior tibial, while the anterior tibial was free. It has been attributed to occupation, but in von Schroetter's opinion without sufficient warrant. The only theory that seems to appeal to von Schroetter is that the affection is, in some manner as yet unknown, dependent on some nervous influence.

Symptoms are made up of prodromata extending through a period of years, as many as twelve, and of such phenomena as depend upon interference with local circulation. Individuals thus afflicted complain for years of pains in the leg or arm which are generally thought to be either rheumatoid or neuralgic, and are likely to be treated as such, yet without benefit.

After a time perversions of sensation occur (paræsthesiæ), as formication, numbness, etc. At first the pains are lessened or disappear when the extremity is at rest, but at length grow extreme, and on use of the affected member become intolerable. As the obstruction to circulation increases movement becomes difficult and the extremity feels heavy, so that the patient favours the limb so far as possible and may actually walk lame.

When at last the artery is wholly occluded areas of gangrene make their appearance. These may be superficial or may invade a toe or the whole foot, and show a tendency to spread rapidly upward. The extremity now looks either pale or livid and feels cold and lifeless. Unless the gangrenous area is removed by the surgeon septic phenomena may develop and lead to a fatal termination of the case. The course of the disease is progressive, and the termination is usually or invariably fatal in the course of years.

The **diagnosis** is surrounded by considerable difficulty, particularly in the prodromal stage. The pains are likely to be considered rheumatic or simply neuralgic, and cannot very well be correctly interpreted before there is evidence of rigidity of and want of pulsation in the arteries.

Obliterating endarteritis is to be distinguished from arteriosclerosis mainly by the age of the patient, since it has been observed most frequently between twenty and thirty, next between forty and fifty, and arteriosclerosis occurs most often past fifty. The disease is likely to be localized, while evidence of vascular degeneration is usually more wide-spread. Moreover, arteriosclerosis, although it may cause gangrene, does so far less constantly than does the endarteritis, and then usually in persons who present well-marked evidence of the arterial change in both legs.

Reynaud's disease, for which the endarteritis may be mistaken, occurs most frequently in children and young adults, especially in females, sets in abruptly, and the dead feeling of the fingers of both hands is attended with slight anæsthesia. Moreover, the condition is due to a cramplike constriction of the vessels, and is not attended with rigidity and pulselessness of the vessels.

The **prognosis** is unfavourable, since the affection is progressive.

Treatment is of a necessity symptomatic and restricted to such measures as may alleviate suffering. The occurrence of gangrene calls for surgical interference.

V. PERIARTERITIS NODOSA. SYN.: CONGENITAL ANEURYSM

The very remarkable and rare affection which bears the above titles was first adequately described by Kussmaul and Maier in 1866, although it appears that Rokitansky in 1852, and possibly Pelletan in 1810, observed each a single case (von Schroetter). The designation Periarteritis Nodosa was bestowed upon it by Kussmaul because of his conception of the process as an inflammation originating in the adventitia. The term Congenital Aneurysm is applied to it because it has been thought to be due to congenital weakness of the arterial coats (Eppinger), leading eventually to the development of multiple aneurysms. According to von Schroetter, only thirteen authentic cases have been reported.

Morbid Anatomy.—The affected artery is studded with nodular thickenings of a whitish colour and of variable size, from that of a pin's head to a pea, which are due to circumscribed fibrous thickening of the intima with cellular infiltration of the adventitia and media. The lumen of the vessel may be narrowed or the weakening of its coats may lead to circumscribed dilatations—i. e., multiple aneurysms. These may reach such numbers as to be uncountable. The disease affects arteries of medium calibre, and is found with special frequency in the arteries of the muscles and viscera, as the heart, intestines, spleen, liver, and kidneys, and also of the skin.

Its **etiology** is entirely unknown, but inasmuch as the clinical picture is very like that of sepsis or an infection it may have some such origin (Romberg). The disease appears to attack both sexes about equally and to occur between the ages of twenty and fifty-two (Osler).

Symptoms.—The most striking features of the affection are weakness, rapidly progressing anæmia, and rapidity of the pulse out of all proportion to the temperature. Fever may be present in the beginning, but is of moderate height, and tends to ultimately disappear. There is pain in the muscles which may eventually show atrophic changes and paralysis. Digestive disturbances are present, as anorexia, thirst, and vomiting, and there may be constipation or diarrhœa. There may be albuminuria and casts, and when the arteries of the abdominal organs are affected there is severe epigastric distress. Hæmorrhages from the bowel may also be observed (Romberg) in cases in which the arteries of the intestines are the seat of the disease.

The course of the malady is progressive as a rule, and a fatal termination occurs in from six weeks to three months. Very exceptionally, however, recovery may ensue.

Diagnosis is impossible unless the nodular thickenings can be felt along the course of peripheral arteries or such as situated within the abdomen are yet accessible to palpation. In suspected cases a nodule may be excised and subjected to microscopic examination.

Prognosis is unfavourable, although recovery does not appear to be impossible.

Treatment is purely symptomatic and is limited to attempts to alleviate suffering, build up strength, and check or overcome the destruction of the blood.

VI. STENOSIS OF THE AORTA AND PULMONARY ARTERY

Stenosis of the Aorta may be Congenital or Acquired.—In the former variety the narrowing is situated at the isthmus and may be caused by a too early closure of Botalli's duct and consequent failure of the descending aorta to receive the amount of blood necessary for its proper development or expansion, or a membrane may be stretched across the vessel at the isthmus, having at its centre an opening through which the stream of blood must pass.

Acquired stenosis may be caused by a fibrous band that constricts the aorta at some point within the mediastinum, or it may be compressed by a tumour. Such conditions are, however, rare as regards the arch, since this portion of the aorta is capable of successfully withstanding encroachment upon it by new growths (Romberg).

In the chapter on Dextrocardia is mentioned the case of a child in whom the rotation and displacement of the heart had caused the superior vena cava to be stretched tightly across the aorta and constrict its lumen. I have also in the chapter on Aortic Regurgitation mentioned the case of a man whose ascending aorta was greatly narrowed by a ring of fibrous tissue that completely encircled the vessel and had induced relative insufficiency of the valve.

Symptoms depend upon the degree and seat of the stenosis. In the *congenital form* collateral circulation may become established through the intercostal arteries, the internal mammary, or arteries in the integument and muscles of the back. If such side channels are sufficient there may be no obvious hindrance to the blood-supply of the lower parts of the body, and no untoward effects are experienced.

Romberg mentions a case observed by him in which the arteries of the back provided a means of maintaining the circulation below the point of stenosis, and in which he detected a loud vascular bruit on the posterior aspect of the trunk between the vertebral column and right scapula. The murmur was attributed by him to dilatation of the arteries at that point.

In the *acquired form* narrowing of the aorta is likely to occasion compensatory hypertrophy of the left ventricle and possibly also incompetence of the aortic valve, as in my case. The ultimate effects are those of cardiac inadequacy. In cases in which relative insufficiency does not occur, but the stenosis leads to left-ventricle hypertrophy, the clinical history is likely to be that of narrowing of the ostium or of the disease which causes the constriction of the aorta.

The **diagnosis** is very difficult as a rule, and may be impossible. One may recognise the signs of obstruction to outflow from the ventricle, but may not be able to determine its real nature. The detection of a mediastinal tumour or of chronic fibrous mediastinitis, together with the signs of obstruction—i. e., a systolic bruit over the course of the aortic arch with *accentuation* of the aortic second tone and left-ventricle hypertrophy—might lead to a correct diagnosis. This would be strengthened if as time went on evidence of regurgitation should appear.

Congenital narrowing of the isthmus might be diagnosed if one were to discover compensatory dilatation of the arteries by which collateral flow is established together with hypertrophy of the left ventricle.

Prognosis depends upon the cause and degree of the stenosis, the effects on the heart, and in congenital cases the completeness of collateral circulation. The general health may not be seriously influenced, or the heart may suffer in its integrity, and death be ultimately brought about through cardiac inadequacy. In a few cases the prognosis may be that of the etiological condition.

Treatment is to be addressed to obviating so far as possible the injurious consequences of the acquired stenosis. We can do nothing towards removing the cause.

Stenosis of the Pulmonary Artery is acquired, and is a relatively infrequent condition. It may be due to constriction by a fibrous band, to compression by an aortic aneurysm and a few other conditions, of which isolated examples have been reported. Thus Romberg states that Litten found stenosis of the pulmonary artery from an " echinococcus embolus," while Gerhardt discovered a case of slight compression of the vessel by the left auricle in consequence of this having become distended by a clot. C. O. Weber, cited by von Schroetter, observed pronounced narrowing of this artery by a bean-shaped gumma in its wall. One of the branches of the artery may be constricted through retraction of the lung in interstitial pneumonia.

Symptoms are confined in the main to the secondary effects on the right ventricle or to congestion of the lung back of the seat of stenosis when this is situated within the lung at a distance from the bifurcation.

If the obstruction is in the main trunk or in a branch sufficiently close to the main stem, the right ventricle undergoes hypertrophy and perhaps dilatation with consequent turgescence of the veins of the aortic system and corresponding feebleness of the pulse. It may even lead to relative incompetence of the pulmonary valve with its evil consequences.

Diagnosis is attended with great difficulty, and is likely to be impossible. It must depend upon the recognition of right-ventricle hypertrophy for which no other cause can be determined, or on this with a systolic murmur in the pulmonic area together with *intensification*, instead of diminution of the second tone, as is the case in stenosis of the pulmonic ostium. Systolic pulsation in the situation of the trunk of the artery—i. e., in the second left intercostal space close to the sternum—together with dulness in this area, would greatly strengthen the other signs just mentioned (Romberg).

The **prognosis** is determined by the nature and degree of secondary disturbance. It is of necessity more or less unfavourable.

Treatment is entirely symptomatic, and, as in stenosis of the aorta, must aim at maintaining cardiac adequacy, since the cause cannot be removed.

VII. CONGENITAL SMALLNESS OF THE ARTERIES

This state of the aorta and arterial system was studied by Virchow, who pointed out its association with chlorosis. Not only are the vessels of small calibre, but their coats are thin and delicate, rendering them particularly liable to rupture, and they are abnormally elastic. In extreme cases the lumen of the arteries may be reduced to a third of the normal (Romberg).

The heart is also abnormally small, the genitalia are likely to remain undeveloped, and the individuals are small and delicate in appearance. This is especially true of those who present the chlorosis spoken of. In other not pronounced cases of arterial hypoplasia there may be nothing in the appearance and no lack of body development to suggest its existence.

Symptoms of this condition as such cannot be said to exist. The heart, by reason of its smallness, is weakened in its resistance, and is more than usually liable to infection (Romberg), and indeed general vigour and resistance may be said to be below par. This is readily comprehensible in cases characterized by chlorosis. The hypoplasia is found more often among females than males.

Fraentzel was of the opinion that congenital narrowness of the arteries predisposed to hypertrophy and dilatation of the left ventricle, and in support of his view cited instances of the kind in young recruits. Romberg, however, thinks the clinical picture drawn by Fraentzel is to be interpreted as the result of prematurely developed arteriosclerosis. This is favoured possibly by the smallness of the arterial system; and yet, as a matter of fact, such arterial degeneration does not occur with special frequency in the subjects of arterial hypoplasia.

It is worthy of note that rupture of the aorta and dissecting aneurysm are said to occur with relatively greater frequency when it is congenitally narrow. The patients are also said to bleed more easily than normal persons owing to the thinness of the vascular coats.

Diagnosis of arterial hypoplasia is difficult to make with certainty. It may be considered as possibly present when palpation of the large cervical arteries and percussion of the heart show what seems to be abnormal smallness of the same, and when, in addition, the individual is poorly developed, chlorotic, and possesses deformity or an infantile state of the genital organs. It is possible that an expert in the use of the fluoroscope might be able to recognise that in a given case the heart and large vessels were abnormally undersized.

Prognosis.—Congenital narrowness of the arteries affects life prospect only when the hypoplasia is considerable and is attended with chlorosis. In such cases there is danger of some of the consequences that have already been considered.

Treatment cannot affect the underlying condition, and is therefore limited to attempts at relieving or modifying such effects as may result.

CHAPTER XXXIV

ANEURYSM OF THE THORACIC AORTA

ANEURYSMS have been the object of interested study for several centuries both to anatomists and clinicians. The names of many celebrated men are connected with the history of this arterial disease, and, as might be expected, they were at first the names of anatomists who studied the subject mainly on the dead body. Methods of diagnosis were crude and, very naturally, not equal to the discovery of such obscure affections as intrathoracic aneurysm. Nevertheless it is worthy of record that Vesalius made a diagnosis of aortic aneurysm in 1567. Malpighi and Morgagni wrote on the subject and added to the facts concerning it. There has been scarcely an author of note since who has not attempted to add to our knowledge on the subject, and to some of them the profession is greatly indebted. Lancisi, Scarpa, Corvisart, Hodgson, Stokes, and in our own time Eppinger and Thoma, are names that are intimately linked with the history of aneurysm.

In this chapter it is proposed to deal exclusively with the disease as it affects the aorta within the thorax, a condition that possesses peculiar interest for the physician. Aneurysms of peripheral arteries belong to the province of the surgeon and hence are left to surgical works for consideration.

Morbid Anatomy.—An aneurysm is a circumscribed dilatation of an artery; and as such must be distinguished from the uniform widening of an artery, which results from sclerosis. The three main divisions that are made of aneurysms are, (A) true, (B) dissecting, (C) false. By false aneurysm is meant a circumscribed collection of blood that has escaped from an artery into the surrounding tissues, hence a hæmatoma. The walls of the tumour are not composed of the arterial coats, and therefore, according to von Schroetter, it should not have the term aneurysm applied to it at all. A dissecting aneurysm is one in which the stream of blood penetrates through a rent in the intima into the parts beneath, and burrowing its way either between the inner and middle coats or in the layers of the media, thus dissects up the intima for a variable distance. In some instances the blood-current again breaks through the intima and becomes reunited with the main stream. This condition may be of long standing and is scarcely open to recognition.

True aneurysm is therefore the condition in which are fulfilled the requirements stated in the definition. The two subdivisions of this form of tumour which best meet the facts as observed by the clinician are (1) fusiform and (2) sacculated aneurysm. By the former is meant a localized dilatation of an artery involving its entire circumference; while by sacculated is meant a dilatation limited to one side, and hence involving but a portion of its circumference.

Aneurysm of the aorta may be either fusiform or saccular, but the latter is the more common. All three coats are involved in the bulging but are not all retained in the wall of the aneurysm. The intima extends into the sac to a greater or less distance, but is then lost. The portion that persists usually presents the changes of arteriosclerosis, as does also the inner coat of the aorta, in the neighbourhood of the tumour.

The media is also involved in the destructive process which has favoured the formation of the aneurysm. Its muscular fibres are degenerated or wholly lost and its elastic elements show signs of granular change. In places, the middle coat may be entirely destroyed; and when such is the case, together with loss of the intima, the wall of the sac is composed solely of the adventitia. This latter is also thickened and infiltrated with inflammatory products.

The pouch which has thus been formed communicates with the lumen of the aorta by an opening of variable size, but almost always smaller than is the calibre of the sac. The interior of the aneurysm is apt to be lined by coagula in the form of layers of a whitish colour. The most internal of these lamina is likely to be reddish and soft, while the more deeply situated layers are firm as well as white. The degree of thrombus formation within the aneurysm is variable, but does not usually fill up its lumen.

Exceptionally, however, when the sac is not very large and its opening into the channel of the aorta is small, its cavity may be entirely filled with coagula so as to obliterate the sac. The innermost layer of fibrine then forms a firm wall nearly on a level with the intima. Its surface is apt to be rough and calcified. Although an aneurysm may in this manner undergo spontaneous arrest, still the degeneration of the arterial coats which led originally to the formation of that aneurysm is likely to favour the development of others, so that multiple aneurysms are not at all uncommon.

Aortic aneurysms differ much in shape and size. Thus a sacculated aneurysm may have other sacs springing from its walls so that the tumour presents an irregular outline. In size the sac may vary from that of a small nut all the way to that of a man's head. Aneurysms may be situated at any point along the course of the aorta from just above the ring to the termination of the abdominal portion.

The disastrous effects of aortic aneurysm are not confined to the vessel, but consist of all the changes in structure and position of neighbouring organs produced by pressure of the sac. The nature and extent of these secondary pressure effects are determined by the situation as well as the size of the aneurysm. Aneurysms involving the sinuses of Valsalva are not apt to attain much size, yet their influence on the heart is very disastrous and they are especially liable to rupture into the pericardium, causing sudden death.

Aneurysms of the arch displace the heart downward (Fig. 109) and it may be forward or to the left, but they rarely occasion hypertrophy of the left ventricle unless the aortic valves have been rendered incompetent. The latter condition is likely to result when the sac springs from the ascending or transverse arch and has attained great size. I recall a man whom I treated for months for aortic regurgitation without suspecting the existence of an aneurysm until quite suddenly signs of pressure on the left lung arose. Even then other signs of the aneurysm were not at all distinct, yet were of such a kind as to render its presence certain.

Other effects of aortic aneurysm than those already mentioned will be left for consideration under Symptoms.

Etiology.—Arteriosclerosis has long been recognised as predisposing to the development of aneurysm. It is objected by Eppinger that the changes of sclerosis tend to render the vessel more rather than less resisting, an objection that is also recognised by Thoma. Consequently the latter points out that aneurysm is likely to develop during the time of primary degeneration and weakness of the media, before compensatory thickening of the inner coat has taken place. This will be referred to again.

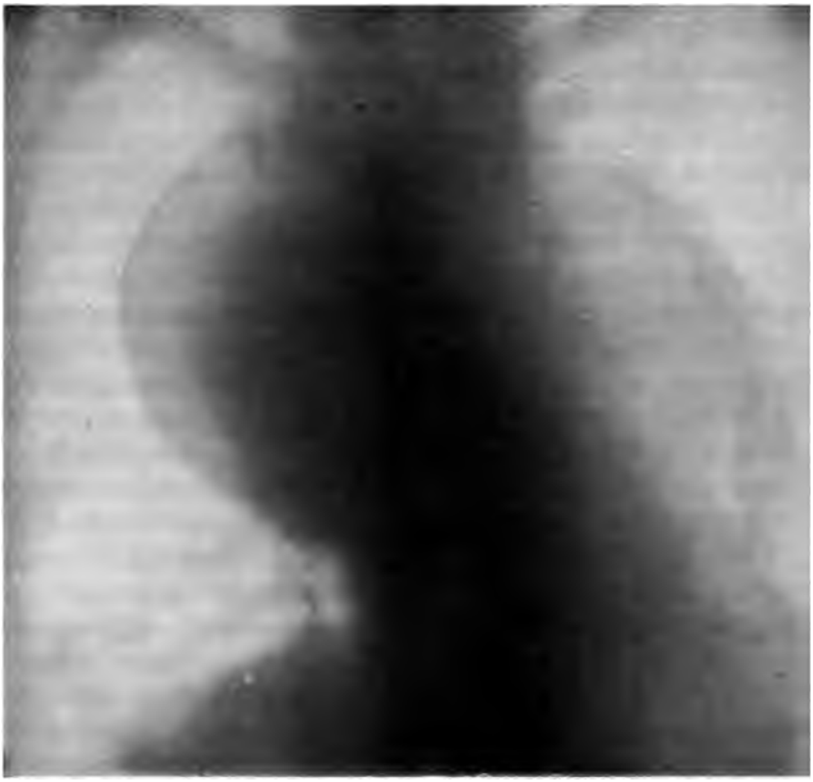


FIG. 109.—SKIAGRAPH SHOWING ANEURYSM OF AORTA WITH DISPLACEMENT OF THE HEART DOWNWARD AND TO THE LEFT.

Syphilis is an undoubted factor in the causation of aortic aneurysm, and yet wide differences exist in the opinions of writers concerning the frequency of its relation to this form of vascular disease. The extremes are represented by M. Schmidt, who finds syphilis present in 29 per cent of cases, and Drummond, who believes that lues is responsible for aortic aneurysm in every instance—i. e., 100 per cent. My experience leads me to look upon

Drummond's opinion as too extreme, and to accept Gerhardt's 53 per cent as much nearer the truth.

Age is a predisposing factor of great importance, since aneurysm of the thoracic aorta is undoubtedly more frequent after than before the fortieth year. The decade of life in which it is most common is still unsettled, and figures differ all the way from the fourth decade (Crisp) to the seventh (Juda, Barsdorff). Thoma's notion is that persons are especially liable to the development of aortic aneurysm at or about the age of forty, in consequence of diminished resistance of the vascular coats at this time. There is, he thinks, a period of about a year at this age when the weakness of the media has not yet become offset by growth of connective tissue in the intima, and during which time the coats of the vessel are therefore liable to yield to excessive blood-pressure at one or more points resulting in future aneurysm.

I have under observation at the present writing a muscular man of forty-four who gives no history or signs of previous syphilis, but who has been a more than usually active, energetic business manager in a line of work that necessitated much physical exertion. This patient suffers from symptoms which, together with stiff arteries and suggestive but not conclusive physical signs, are yet suspicious of fusiform aneurysm of the arch. The age of this person, his occupation, and the state of his arteries, are all, from an etiological standpoint, highly suggestive and strengthen the conclusion to be drawn from the clinical findings.

Sex is likewise a predisposing element in the class of cases now under consideration. Men are without doubt far more liable to aneurysm than are members of the gentler sex. Thus of a total of 425 cases of aortic aneurysm analyzed by Hodgson, Bizot, and Browne, and cited by Gibson, 380 occurred in males and only 45 in females. This striking preponderance of men is not to be attributed to any quality inherent in sex *per se* as inferior vascular resistance on the part of men, but to the greater liability of males to all those factors which favour the development of arteriosclerosis as well as their greater exposure to syphilis and conditions of vascular strain which, acting in conjunction with vascular degeneration, are known to predispose to the occurrence of aneurysm. Sex is therefore only incidentally of etiological influence.

Two other factors that are mentioned as predisposing to aortic aneurysm are the abuse of alcohol and occupations which necessitate vascular overstrain. Both are recognised causes of arteriosclerosis, and as such operate in the production of aneurysm; but, in addition, overwork subjects the aorta to strain at a period of life when, according to Thoma's view, the vessel-wall is least able to endure high intravascular pressure. Such influences are independent of sex, and yet are some of the things which render men more liable to aneurysm than are women.

Race, which is said to exert a certain degree of influence, can scarcely be separated from conditions of work, habits, etc., to which peoples of some countries are especially subjected. Thus aortic aneurysm is particularly frequent in England. The English appear to be more than commonly subject to arterial degeneration, and this fact, acting in conjunction with heavy toil in the manifold workshops of their country, probably accounts for the relatively great frequency of thoracic aneurysm among them. Traumatism cannot be ignored in the production of aneurysm of peripheral arteries, and probably also of the abdominal aorta, but it is difficult to see how injury can have direct etiological relation to aneurysm of that portion of the vessel which is situated deeply within the thorax and is protected by its bony walls. It certainly could only act in connection with already existing degeneration of the media. If under such conditions trauma—e. g., a fall from a height—were to suddenly raise blood-pressure, it might possibly induce laceration of the middle coat and thus be an indirect cause of aneurysm.

The influence of malignant endocarditis in the causation of so-called mycotic aneurysms has been emphasized by Eppinger and is generally recognised. Aneurysms of this origin are usually located in peripheral vessels, and yet it is possible for such aneurysm to be aortic, as shown by the case mentioned by Osler as having occurred in the Montreal General Hospital. In this case there were, in addition to ulcerative endocarditis, four saccular dilatations of the aorta, one large and three small ones. Embolism may also be a cause of aneurysm of the arch as well as of other arteries, as shown by reported instances in which the lodgment of an embolus on the intima of the ascending aorta has been discovered and was associated with circumscribed inflammatory change. Osler thinks it possible for such an embolus, if consisting of a calcareous plate, to lacerate the intima and thus initiate aneurysm.

Lastly, Osler believes there may be an inherent weakness of the vascular coats which predisposes individuals to aneurysm, and cites the instance of Dr. Thomas King Chambers, who, after having had one of the left popliteal artery, and eleven years afterward another in the right leg, finally developed "aneurysms of the carotid arteries."

Symptoms.—Cases of aortic aneurysm may be divided into three groups:

(1) Those in which the tumour fails to declare its presence by either subjective or objective symptoms. Such aneurysms are usually small and are only discovered at the necropsy, when they may be found associated with some other clinically recognisable disease or as the cause of unexpected death through rupture. When the sac is situated just above the aortic ring, it is very apt to rupture into the pericardium. This was found to be the case in 75 out of 289 cases of rupture from a total of 953 instances of aortic aneurysm analyzed by Hare and Holder.

(2) Aneurysms which occasion subjective symptoms as the leading feature of the case. In the majority of cases objective signs are also present, but often of so indefinite a character as to furnish no clear information concerning the nature of the tumour occasioning pressure. Such cases belong to Bramwell's second category. They may be said to correspond also to Broadbent's subdivision of cases which occasion symptoms but not signs of aneurysm.

(3) Aneurysms which produce distinctive physical signs. These are generally united with symptoms of greater or less severity, but the objective manifestations of the disease are sufficiently pronounced to warrant their classification in a separate group.

Aortic aneurysms may also be classified according to their situation—e. g., of the ascending, of the transverse, and of the descending portion of the arch, etc. Indeed, one cannot deal with this subject adequately and clearly without describing the features distinctive of aneurysm in the several locations. There are, however, certain general features shared to a greater or less extent by all aneurysms, whatever their position along the course of the thoracic aorta, and hence these will be considered first.

Such symptoms are the result of pressure, and hence it is plain that variations in pressure phenomena are determined by several factors, as the size of the sac and the direction in which it grows, as well as the portion of the aorta from which it springs. Moreover, aneurysms are liable to change their direction of growth, so that symptoms sometimes differ in character and intensity from time to time. Indeed it may be said that such lack of constancy is generally regarded as one of the points of distinction in favour of vascular as against solid tumours.

Pain is one of the earliest and most constant symptoms of thoracic aneurysm. Its nature and severity depend upon the direction in which the sac develops. If this is towards the surface of the chest, or, as Walshe termed it, "centrifugal," pain appears earlier, is more constant, and more like what is called neuralgic, is sharp and lancinating or dull and aching, and is not infrequently described as boring, grinding, cutting, burning, etc. As it is due to pressure upon the intercostal nerves or branches of the brachial plexus, it is apt to radiate along the lines of these nerves, hence around the chest, up into the side of the neck, down the arm, etc.

As a rule, the pain is confined to nerves connected with the first, second, third, and fourth spinal segments (Head), and is associated with tender areas in the upper part of the thorax at either side, but especially at left of the sternum. Such areas of tenderness are not characteristic of aortic aneurysm, however, for they may be symptomatic of various diseases of the intrathoracic viscera. The pain of aneurysm is apt to be very constant, and in this regard indicative or suggestive of tumour rather than of any other disease not occasioning pressure.

If the sac grows inward towards the more yielding and less sensitive structures, it is not so apt to give rise to such severe pain, and hence this symptom is likely to be overshadowed by some other more distressing symptom, as dyspnœa or cough. The character of the pain, too, when this is experienced, is apt to be more dull and oppressive, and does not radiate so widely in the wall of the chest or the upper extremity. Although the pain of aneurysm is apt to be constant, it is liable to paroxysmal exacerbations which greatly increase the suffering. Pain is also apt to be influenced somewhat by the position of the patient's body. For example, it is apt to be intensified when the patient lies in such a manner as to permit the sac to gravitate or press more strongly upon the irritated and painful nerve. *Per contra,* suffering is lessened by attitudes which allow the sac to fall away from the part previously pressed upon. Such postural variations in the pain are not often marked, but are seen sufficiently often to merit attention.

It is stated also that in some cases the pain is what is known as intrinsic, by which is meant pain experienced in the sac itself or in the aorta either from acute aortitis or from internal pressure. Pain of this origin is evoked or aggravated by increase of blood-pressure, and is dull or aching in character and substernal in location. It is likely to be lessened whenever vascular tension is lowered. Extrinsic pain or that due to pressure may disappear after the structure subjected to pressure has been destroyed—e. g., after the bony wall has been eroded and the tumour is permitted to grow without the restraint of rigid structures. I recall the instance of an enormous aneurysm which had thus penetrated the chest-wall and was covered only by a thin layer of skin, and in which case the man made no complaint of pain whatever.

Dyspnœa is another very common symptom of aortic aneurysm, but varies much in severity. It is of course most pronounced when the growth of the tumour is inward and pressure is exerted on the trachea, large bronchi, or lungs. Very distressing paroxysms of dyspnœa are occasioned by irritation, not paralysis, of one of the recurrent laryngeal nerves, more often the left, and are due to laryngeal spasm. There is apt to be an associated feeling of constriction and perhaps pain in the side of the throat. In a case of the kind coming under my observation, the man felt the painful sense of constriction in the side of the neck corresponding with the recurrent nerve affected, and described the sensation as beginning in the left side of the larynx and running thence along the side to the back of the neck.

I have quite recently seen, in consultation with Dr. Gorgas, a man of fifty with aneurysm of the ascending and transverse arch whose dyspnœa was extreme, and compelled him to maintain the right lateral decubitus. Change of position induced a paroxysm of air-hunger accompanied by uncontrollable coughing. It was impossible for him to rest on the left side, or indeed to lie back against the pillows. In this respect his dyspnœa corresponded with what appears to be a quite common experience—i. e., the influence of posture over the intensity of the dyspnœa and of change of position in evoking a paroxysm of respiratory difficulty that is very like an asthmatic attack. The subsequent history of this case is interesting and instructive. Having received a hopeless prognosis from his medical advisers, he resorted to a Christian Science healer. Owing to a coincident change in direction of pressure, his sufferings abated and he again got about, the improvement being attributed by himself and family to this treatment. After a respite from suffering of several weeks, his former symptoms recurred with aggravated intensity and shortly thereafter the man died.

Dr. Gorgas made an autopsy and discovered an enormous sac that had not only produced pressure on the right lung and surrounding structures, but had caused the erosion of several dorsal vertebræ.

Difficulty of breathing is very apt to be accompanied by stridor, which may be so intense as to be audible at a distance and occasion pronounced fremitus. This stridulous respiration is due to constriction of the trachea or of a bronchus and consequent interference with the expulsion of mucus accumulated behind the point of compression.

Cough is a very common symptom in cases of thoracic aneurysm, but is variable in both frequency and severity. In some cases it is so distressing as to rob the patient of needful rest, and when once excited is so prolonged and intractable as to necessitate absolute repose in a given position and even require the free use of morphine. When due to pressure upon the trachea, as occurs most frequently in cases of aneurysm of the transverse arch, the cough is apt to possess a harsh strident character; that by Wyllie was likened to the note of a gander, and hence is known as the "goose cough." In some instances it may be of a toneless, muffled character, probably in consequence of paralysis of a vocal chord. The causes of cough are various, as (A) reflex irritation from pressure on the vagus or recurrent laryngeal nerve, (B) compression of trachea or bronchus, (C) direct impingement on the lung with resulting retention of secretions or with an actively destructive process.

Expectoration is apt to be associated with cough, and may consist of mucus and serum, muco-pus, and in cases of pulmonary gangrene, of offensive material characteristic of this affection.

Hæmoptysis is by no means uncommon in cases of aortic aneurysm, in which event the blood may come from granulations situated on the tracheal mucosa (Osler), from bronchial congestion, or from destruction of a lung, or from the sac itself, what is then known as weeping of the aneurysm. Such hæmoptyses may occur from time to time over a protracted period, even for months.

I vividly recall the instance of a man with unmistakable aneurysm of the upper portion of the descending aorta, whose clinical picture was that of phthisis. The tumour occasioned destructive pressure on the left lung, with pronounced dulness, bronchial respiration, and a multitude of coarse and fine bubbling râles, frequent harassing cough, and copious purulent sputum which was occasionally streaked with blood. In another case of aneurysm similarly situated, pressure was chiefly exerted upon the left bronchus with consequent dyspnœa, cough, and copious râles due to retention, since there was very little expectoration.

Dysphagia is another very frequent subjective symptom, which is occasioned by aneurysms of the transverse and descending portions of the arch, or when a sac situated on the descending aorta exerts pressure upon the œsophagus. The patient not infrequently speaks of the ingesta seeming to stick at a certain point in their passage downward. If the aneurysm is situated low down near the diaphragm it may cause regurgitation of the food. Digestive disorders, properly speaking, do not form a part of the clinical history of thoracic aneurysms. They may be present nevertheless, and are then the result, in part at least, of the stasis within the portal system and its tributaries occasioned by pressure on the great veins in the thorax.

All aneurysms of the arch do not occasion appreciable interference with the flow of blood out of the venous system. When, however, an aneurysm attains considerable size it can scarcely fail to affect circulation by mechanical pressure. One or both of the venæ cavæ may be compressed, and to such a degree that the circulation can only be carried on by means of collateral vessels.

Such a condition is admirably shown in Fig. 110, which is taken from a photograph kindly furnished me by Dr. Emil Beck. This man, aged thirty-seven, was first seen by Dr. Beck in October of 1901, at which time his complaint was of cough, dyspnœa and inability to lie down. He gave a history of syphilis sixteen years

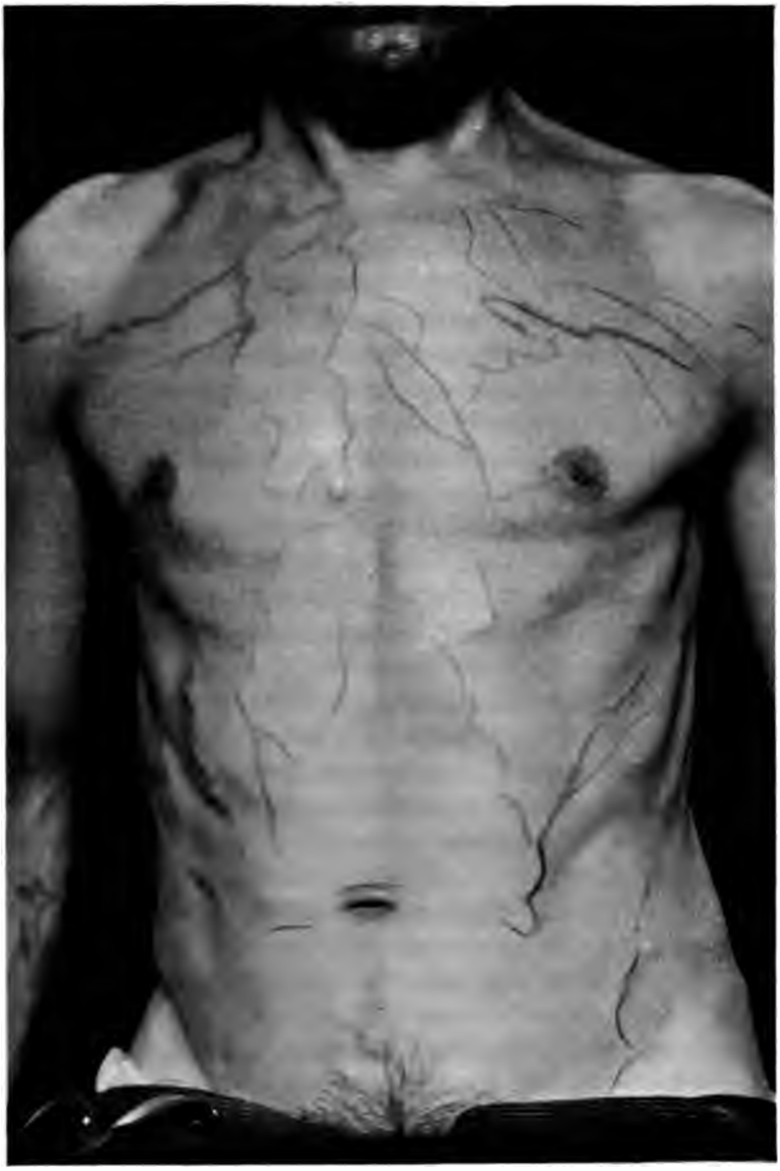


FIG. 110.—DILATATION OF SUPERFICIAL VEINS SECONDARY TO PRESSURE BY ANEURYSM ON VENÆ CAVÆ.

before, for which he received very inadequate treatment. His occupation was that of a metal-polisher, which necessitates the putting forth of considerable strength in pressing the metal against a polishing wheel. Here, then, were two factors both operative in the etiology of aneurysm.

His one initial symptom of breathlessness on exertion developed slowly, and did not necessitate abandonment of work and the sport of playing baseball until nearly a year after it was first noticed. When Dr. Beck examined the patient there was a perceptible fulness of the neck and bulging in the aortic area. This tumour pulsated and gave a systolic bruit. The pulses of the right half of the neck and of the corresponding arm were distinctly smaller than their fellows on the left side. The diagnosis was accordingly made of aneurysm of the ascending and transverse aorta.

Cyanosis and turgescent veins were marked. He was then advised to enter St. Joseph's Hospital, in the service of Dr. Carl Beck, for the purpose of treatment. Rest and iodide of potash did not seem to ameliorate his condition, and he left the hospital.

An aggravation of symptoms and evident increase in the size of the sac led the patient to re-enter, in January, 1902, when he was given hypodermic injections of gelatin (2 per cent in 30 cubic centimetres of normal salt solution) which were administered once a week, subsequently increased to 45 cubic centimetres, until he had received ten such injections in all.

Under this treatment pressure symptoms nearly disappeared, and the patient felt so well that he again left the hospital. Through the courtesy of Dr. E. Beck, I had the opportunity of examining him a number of weeks later. The distention of the superficial veins was then as shown in Fig. 110, while, viewed from the side, there was the evident bulging of the chest shown in Fig. 111. The arteries of the right arm and corresponding half of the neck were manifestly less filled than those on the opposite side. There was a feeble pulsation in the prominent area, and tracheal tugging could be plainly felt.

Percussion elicited an area of flatness having a semicircular outline below and extending from beneath the middle of one clavicle across the upper sternal region to about the same distance on the other side. This area is shown by a shaded area in Fig. 112. Over this area could be heard a dull first tone accompanied by a systolic bruit and succeeded by a loud, ringing second sound.

The area of relative cardic dulness is also shown in Fig. 112,

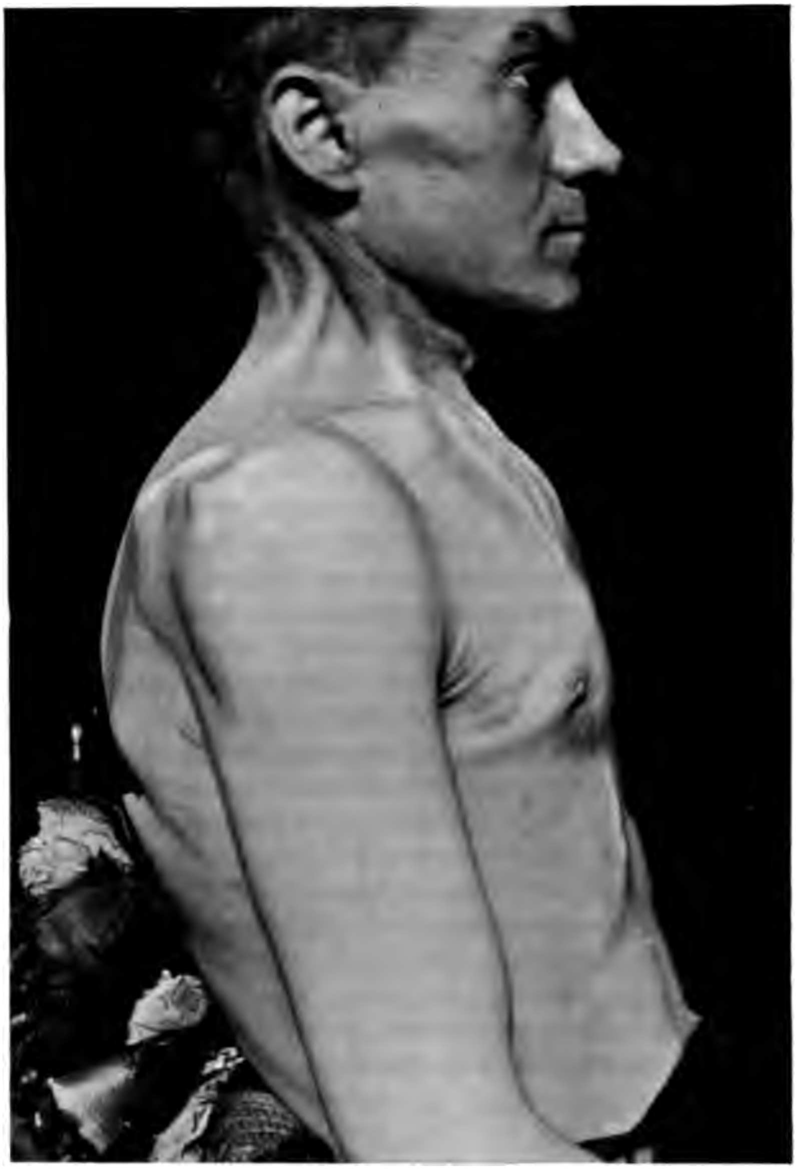


FIG. 111.—PHOTOGRAPH OF CASE OF AORTIC ANEURYSM, SHOWING SLIGHT BULGING OF ANTERIOR CHEST WALL.

and from its position indicates displacement of the heart downward and to the left. Its sounds were clear, but the aortic second was very loud and metallic.

The liver, as indicated by the outline at the bottom of the figure, was evidently engorged as well as probably somewhat depressed, being palpable and having an area of greatly increased flatness.

The man admitted having previously noticed some difficulty in swallowing. He had not experienced pain to any extent, but in the last two weeks had begun to notice some dull pain in the front of the chest at right of the sternum. This, it seemed to me, indicated an increase of pressure upon the parietes, the sac having changed its direction of growth, and hence its pressure, in the weeks following his abandonment of the gelatin injections. It may be stated in addition that the nature of this case was confirmed by an X-ray examination.

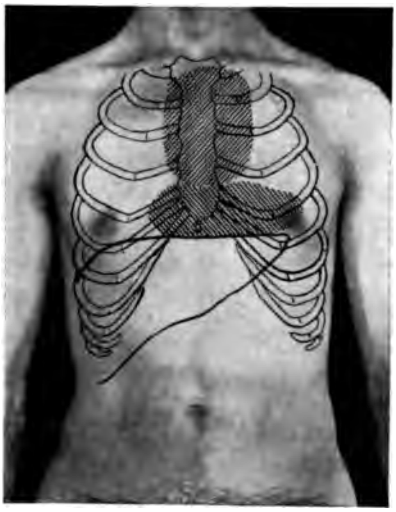
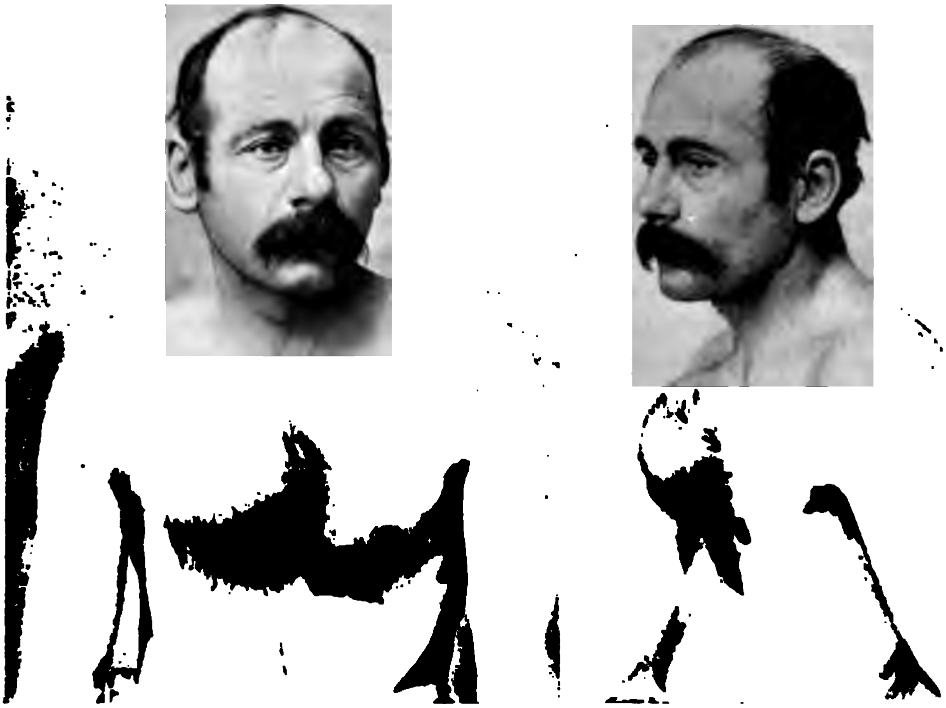


Fig. 112.—Shows Dulness and Liver Outline in Case of Aneurysm (p. 785).

The interference with venous circulation in these cases may not only be declared by turgescence of superficial vessels, but by general or localized œdema. Thus the neck and upper extremities may become dropsical, or the œdema may be limited to one arm and a portion of the thoracic wall. Inequality of the pulses on the two sides is very common, owing to partial obliteration, displacement, or twisting of the great branches given off from the arch. Displacements of the heart occur and the function of the valves, especially the aortic, is quite likely to be seriously interfered with. Pressure effects in detail will be considered in connection with the description of aneurysms in the various situations.

(1) *Aneurysms of the ascending portion of the arch.*—These may be situated close to the aortic ring and involve the sinuses of Valsalva, or they may spring from the convex or concave surface. In the first situation they are apt to be small and to escape detection, first declaring their presence by rupture into the pericardium and death.

If the sac arises from the convex aspect, it is likely to attain great size and exert very obvious pressure effects. If its direction of growth is forward as well as lateral, it produces a pulsating tumour in the second and third interspaces at the right of the sternum, and not infrequently leads to erosion of the bony covering. Aneurysms in this situation may attain truly enormous dimensions, and projecting with only the integument for a covering, necessitate the wearing of a metal shield, lest the tumour be accidentally struck and caused to burst. Figs. 113–115 show an aneurysm in this location which had an external diameter of several inches. I vividly recall another man sent to me by Dr. G. Frank Lydston, who presented a pulsating prominence which occupied the entire præcordia, extending from one nipple to the other, and from the upper border of the second rib to the inferior



FIGS. 113, 114.—SHOWING EXTERNAL TUMOUR IN CASE OF AORTIC ANEURYSM (see Fig. 115).

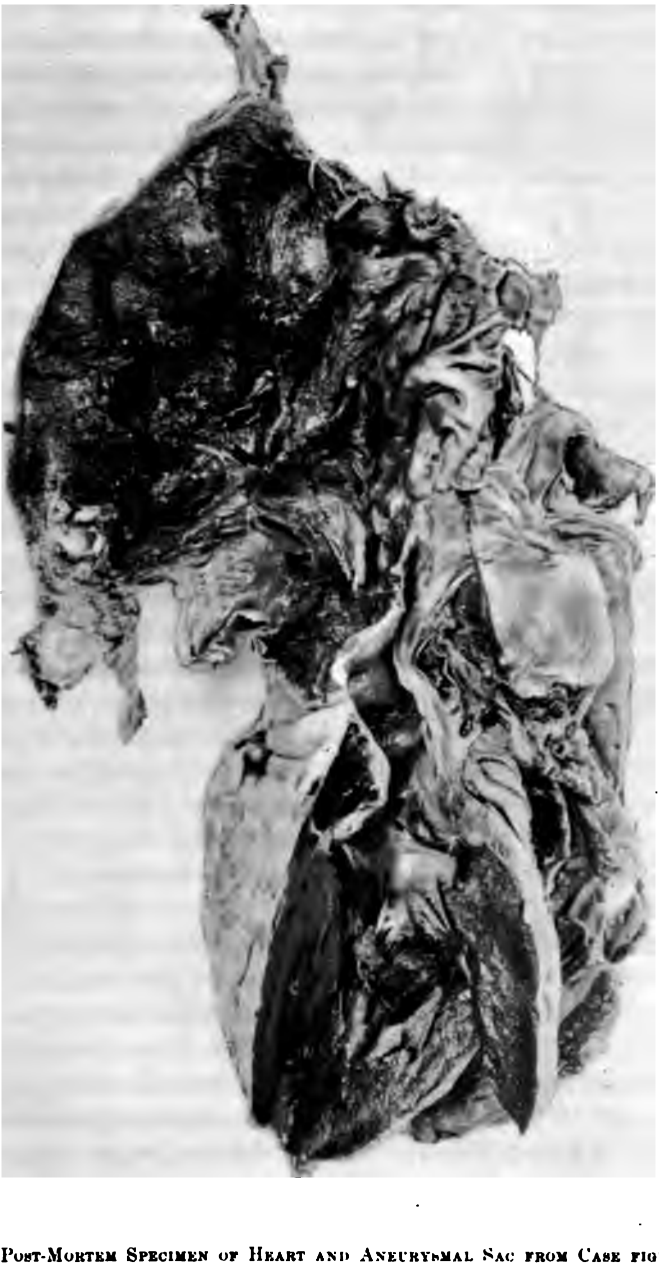


FIG. 115.—POST-MORTEM SPECIMEN OF HEART AND ANEURYSMAL SAC FROM CASE FIGURED IN FIGS. 113 AND 114.

extremity of the sternum. As nearly as could be determined by measurement with calipers, the tumour projected 4 inches at its highest point above the level of the surrounding chest, and its diameter was 7 × 8 inches. In places the enveloping skin was so thin and blue that it seemed on the verge of rupture, and made me actually shudder to touch it. I dared not place a stethoscope upon it firmly enough to auscultate with accuracy, but so far as could be ascertained the dull, distant sounds were not accompanied by murmurs. Where the heart was I could not determine. How this man had been able to thread his way through our crowded streets without receiving a fatal blow on this thin-walled sac I do not know. He was advised to protect it by wearing strapped to his chest a framework or cage of woven wire. He was seen by me but twice, and it is probable that death from external rupture took place not long thereafter.

Aneurysms in this situation may encroach upon the pleural cavity and lung, as witness an instance seen in the Cook County Poor-House in which the necropsy revealed a sac of enormous size that nearly filled the entire right half of the thorax and had caused collapse of the lung, the same as would a massive pleuritic exudate. The side was motionless and moderately enlarged during life, flat and intensely resisting on percussion, with complete absence of cardiac tones and murmurs. Breath-sounds were heard feebly at the summit of the chest behind, close to the spinal column, all of which findings, together with distention of the superficial veins, were held to indicate pressure by a solid tumour rather than aneurysm.

Aneurysms of the convex portion of the ascending aorta are likely to impinge upon the superior vena cava and have been known to rupture into this vessel. They may press also upon the right subclavian vein and occasion passive congestion of the arm and other parts drained by this vein. In some instances the right recurrent laryngeal nerve is subjected to pressure, with consequent paresis of the right vocal cord. The heart is also likely to be crowded downward and to the left, while the aortic valve is apt to be rendered relatively incompetent. The hypertrophy of the left ventricle in such cases is the result of the regurgitation rather than of the aneurysm *per se.*

Aneurysms springing from the concave portion of the ascending aorta may, according to Osler, occasionally give rise to a tumour at the left of the sternum and then occasion great displacement of the heart. There is at the present time in Ward 10 of Cook County Hospital a man who presents such a tumour. It lies in the situation normally occupied by the body of the heart—i. e., between the second and sixth costal cartilages, the left border of the sternum, and 1 inch outside of mamillary line—has a slowly heaving expansile pulsation and gives forth a distinct, harsh double bruit that has replaced the normal cardiac sounds. The heart, as shown by percussion and the location of what appears to be the apex-beat in the seventh interspace midaxillary line, is greatly displaced downward and to the left. Its tones are rather feebly audible in this situation and are accompanied by the same to-and-fro murmur, though less distinctly than on the body of the tumour. Vascular signs of aortic regurgitation are present, and there is an indefinite tracheal tug. There are no signs of pressure on the left recurrent laryngeal nerve, and pressure effects on veins are not present. Without wishing to affirm that this sac arises from the concave aspect of the ascending aorta, I yet incline to the opinion that such is the location, since the incompetence of the aortic valve is not so likely in cases of aneurysm developed from the descending portion of the arch, and were the transverse arch the portion affected, the tumour would be likely to have a different location. This patient has been an inmate of the hospital at various times for the past four years. Occasional dull pain over the seat of the growth and dyspnœa of effort are the only symptoms of which he complains.

(2) *Aneurysms of the transverse arch*, in the same manner as those just considered, produce a variety of effects according to their size and direction of growth. They most frequently develop in a backward direction, and then, when even of small size, occasion pronounced symptoms in consequence of pressure on the trachea and œsophagus, interfering with respiration and deglutition. Paroxysmal cough is a very common symptom and inspiration is attended with stridor.

Growth of the sac forward produces a tumour at the upper part of the sternum and to the right, with absorption of the bony structures. The tumour may occasionally present at the left of the breastbone, but does so so much less commonly than at the right of the median line that, according to Osler, O. A. Browne found it but 4 times out of 35 cases of aneurysm of the transverse arch. These tumours sometimes reach enormous size and fill up the superior mediastinum so that they spread out into both pleural cavities.

Pressure of these aneurysms is exerted on the left recurrent laryngeal nerve and left bronchus, producing such characteristic phenomena that these cases have been described by Dieulafoy as Aneurysms of the Recurrent Type. In this class of cases symptoms vary according to whether the nerve is paralyzed or merely irritated by pressure of the growth. Paralysis of the recurrent nerve is shown by paralysis of the corresponding vocal cord, which, examined laryngoscopically, is seen in a state of cadaveric rigidity.

When the nerve is merely irritated laryngeal spasm is evoked, shown by paroxysmal dyspnœa, lasting from a few minutes to several hours, and causing very great distress. There is also apt to be painful deglutition, and there may be attacks of angina pectoris from pressure on the cardiac branches of the recurrent (Prebble). Pain in swallowing is due to spasm of the muscles of deglutition in the pharynx and gullet. Variations in the quality and power of the voice are observed in these cases, and the left vocal cord may be paralyzed for a transient period.

Mr. M., a school-teacher, aged thirty-two, was referred to me by Dr. Bayard Holmes because of paroxysms of dyspnœa that were thought by the patient to be attacks of asthma. The history was, that a year earlier he had, one evening, without previous warning, been seized with a fit of coughing that was immediately succeeded by difficulty of breathing lasting the greater part of an hour. As he had become chilled the night before on the deck of a steamboat, he had attributed his attack to having taken cold and thought no more about it.

That was not his last attack, however, but during the next few months he experienced several recurrences. During the six months last past his attacks had increased in frequency and intensity. They came on at any time, but more often in the early morning, waking him out of sleep, and lasting from twenty minutes to half an hour. They were accompanied by loud wheezing in the throat or upper part of the chest and gave him the sensation of being strangled. When the attacks subsided he felt as well as ever. In other respects he felt in good health.

It was apparent that this was not the clinical history of bronchial asthma, but was highly suggestive of some organic disease, especially of intermittent pressure. I therefore inquired concerning syphilitic infection and learned that he had had a chancre twelve years before. This fact in connection with his symptoms suggested thoracic aneurysm as the possible cause of pressure on the recurrent laryngeal, and led to minute inquiry regarding pain and cough. With exception of a trifling dry cough to which he paid no attention, he declared he was free from all symptoms except the spasmodic dyspnœa already described.

The results of examination may be briefly stated as follows: The left radial and carotid pulses seemed not quite so full and strong as the right, but the difference was so trifling that I hesitated to accredit my senses lest I might be deceived by my suspicion of aneurysm into recognising an asymmetry that did not actually exist. Likewise I was not able to positively identify any abnormal finding in the investigation of the heart and cervical vessels, but I thought I recognised a slight difference in the intensity of the second tone on the two sides of the neck, that above the left clavicle being somewhat louder and more ringing than at the right. There was certainly no abnormal pulsation or inequality in the vessels to the palpating finger. The heart appeared in its normal position.

Upon examination of the lungs, however, certain abnormalities were at once detected. Over the upper portion of the chest there was a single inspiratory sibilus with each act, the râle seeming to be more pronounced on the left. But the change that was most noteworthy was dulness of the left apex above the clavicle and in the first interspace close to the left edge of the sternum. This impairment of resonance was not intense and yet was distinct. Over this area the breath-sounds were obscured by subcrepitant râles. After several forced inspirations the dulness became less pronounced and the râles partially disappeared.

These findings convinced me that the loss of resonance was due to atelectasis, and that from the history of spasmodic dyspnœa the collapse of this portion of the left upper lobe was probably caused by pressure. From the history of syphilis twelve years before and from the absence of positive signs on the part of the circulatory apparatus, I was led to make an inferential diagnosis of aneurysm of the transverse portion of the arch and consequent irritation of the left recurrent laryngeal nerve.

Laryngoscopic examination was next made, and aside from slight congestion of the left arytenoid cartilage was negative. The patient was then submitted to an X-ray examination with the result that the fluoroscopic screen revealed a pulsating tumour behind the left edge of the sternum, while a skiagraph showed a distinct though small shadow in the same situation.

The diagnosis was thus confirmed and the case was shown to be one of the type just described. It is interesting, furthermore, in two respects: first, on account of the early age (thirty-two years) at which thoracic aneurysm has developed, and second, because it was the peculiar character of the dyspnœic attacks which suggested the possibility of the disease.

The attacks, which in this case were considered asthmatic by the patient, were in reality due to laryngeal spasm; and could the larnyx have been inspected during an attack, the arytenoid cartilages would probably have been found approximated and the left vocal cord occupying the median line. As a matter of fact, an attempt to inspect the larynx during a spasm was made, but the attack was passing off and the laryngoscopic examination was negative.

It is further worthy of note, that paroxysmal dysphagia was not experienced and that pain was never complained of by this man. This accords with the fact that dyspnœa and dysphagia are not necessarily associated in all cases.

Pressure on the left bronchus is another effect of aneurysms of the transverse arch of the type now considered. If the tube is but slightly constricted, the lung becomes retracted only sufficiently to occasion immobility of the side, tympanitic resonance and diminished respiratory sounds. When the bronchus is greatly narrowed the side becomes perceptibly smaller than its fellow, the percussion note is dull, and respiratory sounds are abolished. There may be retention of the secretions with râles, bronchorrhœa, and bronchiectasis—symptoms which, in the Montreal General Hospital, are characterized as “aneurysmal phthisis” (Osler).

Aneurysms of this portion of the arch sometimes occasion pressure on the thoracic duct. If they develop in such a direction as to involve the innominate or carotid artery, the condition is apt to be shown by a symmetry or delay of the pulses on that side. Pressure on the sympathetic is another manifestation of tumours in this situation, and is shown by dilatation and immobility of the pupil when the nerve is irritated, and by contraction when the sympathetic is paralyzed. Tracheal tugging is another result of aneurysm of the transverse arch, as was first shown by Oliver. It

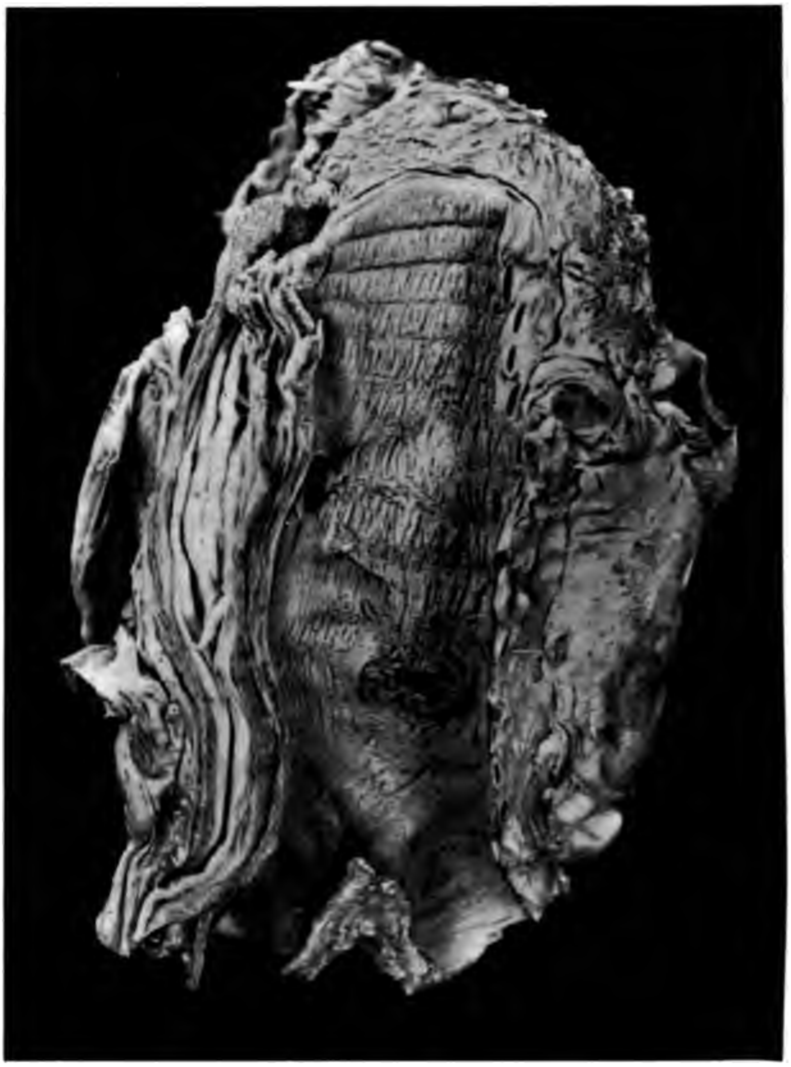


FIG. 116.—TRACHEA FROM CASE OF RUPTURED ANEURYSM, SHOWING POINT OF RUPTURE.

is due to the downward traction of the sac on the trachea at its bifurcation. This sign will be spoken of again at greater length under Palpation. Aneurysms in this situation may rupture into the trachea (Figs. 116, 117).

(3) *Aneurysms of the descending portion of the arch* grow laterally and posteriorly in the majority of instances, and yet it is stated that a tumour of this portion of the vessel may present at the left of the sternum (Sansom, Walshe). Phthisical symptoms are the result of pressure on the left lung or bronchus, dysphagia of compression of the gullet, pain from erosion of the dorsal vertebræ

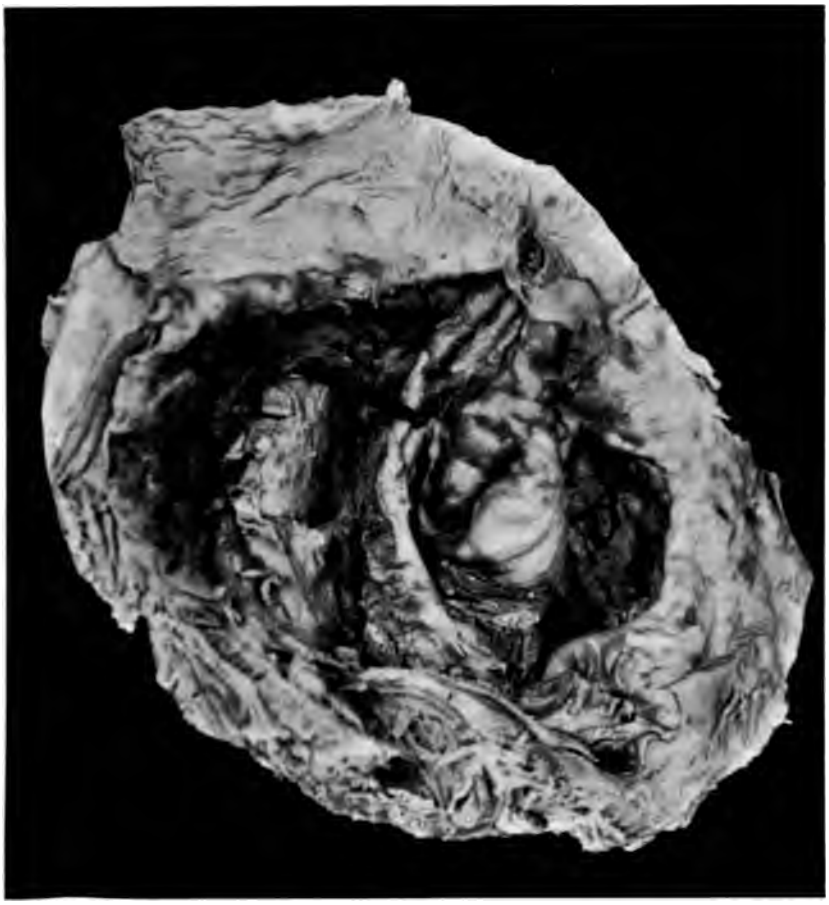


FIG. 117.—OPPOSITE SIDE OF SPECIMEN SHOWN IN FIG. 116. SHOWS INTERIOR OF SAC. PROBE IN OPENING INTO TRACHEA.

(third to sixth), and a tumour in this situation may be the result of backward pressure. Compression of the spinal cord may occasion characteristic effects—e. g., paraplegia.

(4) *Aneurysms of the descending thoracic aorta* are usually located low down near the diaphragm and produce oftentimes very obscure symptoms. In an instance of the kind which I saw with Dr. Bayard Holmes, and which was not recognised as aneurysm, the only complaint was dull pain vaguely felt in the lower zone of the thorax and upper abdominal region. The only thing that could be discovered on examination was an area of impaired resonance and feeble broncho-vesicular breath-sounds in the left infrascapular region, close to the spinal column. From the history of previous illness, that seemed to have been pleuritic, and from the physical findings, this area was erroneously thought to indicate old adhesions. The autopsy, months subsequently, revealed a sac filled with dense coagula pressing on the base of the left lung just above the diaphragm.

Aneurysms in this situation may, as previously stated, cause dysphagia and regurgitation of solid ingesta, but they rarely occasion respiratory embarrassment. Aside from deep-seated pain they are not likely to produce subjective symptoms, and unless, by reason of their size, they give rise to lateral dulness and other signs of pressure on the lung, they are likely to escape recognition.

It should be borne in mind that an aneurysm which in the beginning is confined to one portion of the arch may as time progresses so increase in dimensions as to eventually invade other divisions of the vessel. Thus, a sac at first limited to the transverse arch may in time spread to the ascending portion, or one in this latter situation may at length involve the entire arch; so that both subjective and objective symptoms are very liable to exhibit changes corresponding to the extension of the aneurysm.

I recall the case of a locomotive engineer who was for many months an inmate of the Cook County Hospital in whom such a change took place. His aneurysm at first presented in such a situation that it was believed to implicate the descending portion of the arch. As months went on, however, the tumour grew enormously towards the front, and at the necropsy was found to have involved the entire arch, which had consequently lost all semblance to an arch, being, in fact, but a huge sac from heart to descending aorta.

Physical Signs.—*Inspection.*—In some cases this is wholly negative, minute scrutiny failing to detect signs of pressure, and the general appearance being that of robust health. In other instances, on the contrary, patients look cachectic, and their chests being uncovered present unmistakable evidence of aneurysm. It is not to be supposed that all the signs are present in any one case. Consequently the following are the points to be carefully looked for:

(1) Circumscribed bulging of the chest-wall in the following areas: (A) At the right of the sternum, especially in the second and third intercostal spaces, but also the first, and including the sterno-clavicular articulation; (B) at the upper end of the sternum, including the regions at either side and the fossa jugularis; (C) in the intercostal spaces at left of the breastbone, from clavicle to fourth rib; (D) in the left interscapular region below the level of the fourth dorsal vertebra. These are the areas in which thoracic aneurysm most commonly makes its appearance.

The integument at these points may appear smooth and shining, the prominence being slight, or a tumour of such size may project and have so eroded the overlying structures that the skin is of a dark red or bluish hue, or may have disappeared in spots, leaving the wall of the aneurysm visible.

(2) Signs of interference with the circulation: (A) visible cutaneous capillaries on some portion of the chest, as over the area of bulging; (B) distended, tortuous veins denoting the establishment of collateral circulation in consequence of pressure on some of the great internal veins, as superior vena cava, one of the innominates or subclavians; (C) localized œdema, as of one arm and corresponding half of the neck, or when bilateral, of the upper part of the body, but not of the lower extremities. Walshe speaks of the neck being in some instances so distended and spongy from capillary turgescence as to look "like a collar of flesh."

(3) Pulsation in some abnormal situation—e. g., one of the areas in which bulging may appear; or an exaggeration of a pulsation in a normal situation—e. g., of the cervical arteries, particularly on one and not the other side or in the episternal notch.

(4) Dislocation of the cardiac impulse, in most instances downward and to the left. The organ may, however, be pushed strongly forward against the anterior chest-wall.

(5) Diminution or absence of respiratory movement of one half of the thorax, more often the left, with, in some cases of marked bronchial compression, also retraction of the side. This sign in conjunction with pressure-symptoms is highly suggestive.

(6) Immobility of one pupil, which may be larger than its fellow, but is more often contracted.

(7) Sweating of the head, sometimes unilateral, and by Walshe said to be very profuse in some instances. This is another sign of pressure on the sympathetic, and taken in conjunction with other pressure-symptoms may be of value, but found alone possesses no significance as respects aortic aneurysm.

Palpation is of value chiefly as a means of detecting abnormal pulsation, its extent and character. It is especially likely to give information when employed as bimanual palpation, one hand being pressed firmly against the chest in front and the other behind. In this way, deeply situated pulsation may sometimes be appreciated that otherwise would escape recognition. If a bulging area is perceived to pulsate, one should endeavour to feel the extent, force, and direction of the pulsation. If the tumour is due to aneurysm, it is likely that the pulsation includes the whole area. If this is forcible, so forcible in fact as to equal in this regard the beat of the heart, it is highly suggestive of aneurysm (Balfour). Finally, the pulsation of aneurysm may be slowly heaving and is expansile, and when by palpation this character can be determined, there is no doubt of the nature of the tumour. Pulsation imparted to a solid tumour by a vessel beneath is a simple forward thrust or shock.

In some cases the hand laid upon a tumour due to aneurysm perceives a distinct diastolic shock which succeeds the systolic impulse. This is very characteristic, being due to elastic recoil in the wall of the sac. In some instances a thrill is detected in the bulging area, but in my experience is not at all common, and is of diagnostic aid only in connection with other signs.

Palpation is of value also in the study of the pulse with a view to ascertaining whether or not it is equal and synchronous in corresponding arteries, since when the innominate or the left common carotid and subclavian arteries are implicated, smallness and perhaps retardation or obliteration of the pulse in the arteries of the corresponding half of the neck or arm are likely to be occasioned.

Palpation is of value also in ascertaining displacement of the heart, as well as hepatic congestion due to pressure.

The finger pressed gently into the episternal notch may sometimes detect pulsation of the transverse arch of an abnormal character, or a thrill, as well as the jogging impulse of aneurysm.

The tracheal tug is another phenomenon sometimes elicited by palpation. It is a distinct downward pull of the trachea caused by the impact of the sac against the windpipe at its bifurcation or against a main bronchus, and although feebly present in some other conditions—e. g., free aortic regurgitation—is never so marked as in aneurysm of the transverse arch. To elicit tracheal tugging the examiner instructs the patient to raise his chin so as to strongly extend the neck, whereupon he inserts the tips of his forefingers into the notch between the thyroid and cricoid cartilages and pulls gently upward. If the sign sought for is present, the trachea is felt to be jerked distinctly downward with each cardiac systole. When well marked, this tug cannot be mistaken, but when not pronounced considerable care is required for its detection.

Percussion is a valuable means of diagnosis in cases of aneurysm, especially when there is no visible tumour. Before the sac leads to protrusion of the chest-wall it may occasion retraction of a lung-border or more or less collapse of a lobe, so that dulness in one of the areas in which aneurysm is usually situated may be detected by firm percussion and form an early sign of such tumour. It is especially important to percuss carefully in the right infraclavicular region close to the sternum, since loss of resonance in this location is, together with symptoms and signs of pressure, strongly suggestive of aneurysm. Dulness over the manubrium is not so suggestive as at either side. Percussion is necessary also for the recognition of pressure effects on the lungs and of displacement of the heart.

Auscultation.—Aneurysms do not always produce acoustic phenomena, a statement which applies to some large as well as small ones that are deeply situated. A sac may be filled with coagula, and be thus to all intents and purposes the same as a solid growth, in which event no adventitious sounds are generated and the aneurysm remains silent.

In most instances, however, aneurysms occasion abnormal sounds or bruits which are audible over the sac or in some neighbouring vessel or part to which they are propagated.

There is no auscultatory phenomenon pathognomonic of aneurysm, but certain sounds are more suggestive than are others. The two tones normally heard over one of the great vessels at the base of the neck and in the aortic area are usually altered by the development of aneurysm. Either the systolic or the diastolic may be modified—i. e., intensified, diminished, or impure.

Perhaps the most frequent and striking change is a loud peculiarly ringing quality of the second tone heard over the growth or in one of the cervical arteries but not the others. In some instances such a sound is impure or split, in others it is clear and clanging, while the first is not pure or has been replaced by a murmur of harsh quality. In other cases again the systolic tone is pure and accentuated and the diastolic is accompanied or obscured by a distinct bruit, while in still others there is a double to-and-fro murmur of wide propagation.

Intensification or modification of the normal vascular sounds occurring in immediate proximity to the heart—e. g., in the aortic area, are not so suggestive as are such changes in regions in which they do not normally exist—e. g., the left interscapular region or one side of the neck. Another very valuable auscultatory sign is the propagation of the heart-tones to a much greater distance than normal—e. g., to the outer limit of an infraclavicular region or into an axilla, the lung tissue not being indurated. This condition is essential, for solidification of lung from tuberculosis may lead to wide transmission of the cardiac sounds without aneurysm.

We do not yet understand the conditions which determine changes of one kind and another in the tones heard over an aneurysm. These sounds are probably not generated *de novo* in the wall of the sac, but are merely conducted thither from the heart and are there intensified, reduplicated, or otherwise modified by vibrations set up in the sac-wall or by some other condition that escapes our ken. It may well be that bruits are generated in some cases in the sac itself in consequence of the blood-stream swirling into or out of the sac, but probably the murmur is due in other instances to atheromatous roughening of the aorta between the heart and sac or to insufficiency of the aortic leaflets. This is believed to be the explanation of the double aortic bruit not infrequently heard in aneurysm of the ascending portion of the arch. Indeed, Gibson states that he can recall only three cases in the literature in which such a double bruit was found without associated incompetence of the semilunar valves. It is not strange, therefore, that all possible combinations of tones and murmurs may be heard in cases of intrathoracic aortic aneurysm.

Drummond has called attention to the fact that the pulsation of an aneurysm may be communicated to the trachea and manifested by rhythmical interruption of the expiratory murmur. This is perceived by placing the stethoscope upon the manubrium and auscultating while the patient expires slowly through only one nostril, the other being closed by his finger. This phenomenon is not peculiar to aneurysm, being perceived in health, but is more pronounced.

In some cases an aneurysmal bruit may be plainly heard when the bell of the stethoscope is placed between the patient's teeth, his lips being closed about the instrument. Sansom speaks of having thus been able to detect a systolic murmur, and Dr. E. J. Abbott, of St. Paul, has narrated to me an instance in which the detection of such a tracheal bruit was the only evidence of aneurysm he could discover. In Cook County Hospital at present writing is a man with aneurysm in whom both a systolic and diastolic bruit can thus be loudly heard. The phenomenon is due to the conduction of the murmur to the column of air within the trachea. This sign may be of diagnostic value in cases of small sacs of the transverse arch which are too deeply situated to declare their presence by outward pressure-effects.

Diagnosis.—Under some circumstances the diagnosis of aneurysm of the thoracic aorta may be made almost at a glance, by the discovery of an external tumour displaying the expansile pulsation and other characters of an aneurysm. There are other cases, on the contrary, in which the most painstaking examination fails to positively establish the nature of the malady. Between these two extremes are to be found cases which, although obscure, are yet susceptible of elucidation by minute investigation and by exclusion.

In a suspected case the following points may be considered of diagnostic importance: (1) A history of syphilis years before or of strain, as by occupation, to which some would add chronic alcoholic excess. (2) Age, the patient being at or after the middle period of life. (3) The male sex, since men are vastly more liable to aneurysm. (4) Symptoms indicative of intrathoracic pressure; as, (A) intractable pain of the characters previously described; (B) dyspnœa, especially if influenced by posture; (C) cough of a brazen clang, also evoked or intensified by posture; (D) dysphagia or regurgitation of food. These four symptoms, if all present, form a very strong chain of evidence in favour of an existing aneurysm.

If to the foregoing history and symptoms the following physical signs are added, reasonable doubt can scarcely be entertained: (5) Bulging, even if slight, in some one of the areas in which aneurysm is likely to be present. (6) Dulness in one of these areas even without perceptible bulging. (7) Displacement of the heart, most often downward and to the left. (8) Some of the auscultatory phenomena already described, especially a harsh, aortic systolic bruit with a clanging second sound. If such second tone is split or doubled and is heard most plainly or solely over a dull area or in the cervical arteries, especially if on one side and not on the other, and is accompanied by a diastolic shock, the evidence, taken in connection with pressure-symptoms, may be considered almost conclusive.

Aside from an external tumour having a distinctly expansile pulsation or a diastolic shock, there may be said to be no signs so distinctive as to be pathognomonic. Diagnosis is to be found in the association of several important signs rather than in any one alone. Nevertheless attention may be especially directed to what Balfour considers very trustworthy evidence—namely, a pulsation in an aneurysmal area equal in intensity to the apex-beat, so that there may be said to be two areas of maximum impulse. Even this is not absolute, however; for a kyphoscoliosis has been known to push the convex portion of the aortic arch so strongly against the anterior chest-wall at right of the sternum as to simulate, with respect to the force of its pulsation, a thoracic aneurysm.

Tracheal tugging is a very strong sign of aneurysm of the transverse arch, especially in conjunction with other signs; but as it may be produced by other conditions, it is not infallible.

Differential Diagnosis.—This concerns especially the three following diseases, which taken in order of frequency and importance are: (1) A solid intrathoracic growth—e. g., carcinoma and the varieties of sarcoma; (2) mediastinal abscess; (3) pulsating empyema in close contiguity to the base of the heart.

(1) *Malignant Tumour.*—This disease when situated within the thorax occasions symptoms of pressure so identical in some respects with those of aneurysm that they cannot be distinguished. The chief differential points are to be found, therefore, in the history and physical signs. As a rule, the history is of more rapid growth than in aneurysm, accompanied by more pronounced emaciation and loss of strength. In the physical signs the main differences are found in the character of pulsation, when such exists, and in the auscultatory phenomena.

A solid tumour occasions pulsation which is not expansile, but is a forward impulse, owing to the circumstance that the growth itself does not pulsate, but receives an impulse imparted to it by the aorta or some other artery or by the heart against which the tumour lies. More commonly, however, such a mass possesses no impulse. It must not be forgotten, on the other hand, that when a sac is filled with dense coagula, it is practically also a solid tumour, and hence under such conditions may be also incapable of producing any perceptible pulsation. I recall such an instance in Cook County Hospital. A large, dense, intensely resisting, nonpulsating tumour protruded close to the sternum in the right infraclavicular region. It was, moreover, perfectly silent, and very naturally was for a long time mistaken for a malignant growth. Only after the lapse of time had somewhat altered the size of the sac and permitted vascular sounds to be generated was a correct diagnosis possible.

As regards the sounds audible over a solid tumour, it may be stated that when such are present they are usually clear and unchanged. It is possible, however, for the cardiac or vascular sounds to be modified in consequence of pressure by the growth. Under such circumstances bruits may be generated or the second sound may take on a ringing intensification. It is not likely to be so clanging as is sometimes the case in aneurysm. Moreover, a tumour of the mediastinum which, from its situation and resulting area of dulness, simulates aneurysm of the transverse arch, does not occasion a tracheal tug. Neither is such a solid growth when situated in the area at right of sternum, and hence simulating aneurysm of the ascending aorta, likely to lead to signs of insufficiency of the semilunar valves. It does not change its direction of growth and cause sudden modifications of symptoms, nor is it apt to create asymmetry of the pulses. Finally, in cases of malignant growths there may be history or symptoms of an antecedent tumour elsewhere, or there may be induration of some of the lymph-nodes in axilla or neck which may aid in the correct interpretation of the case.

(2) *Mediastinal Abscess.*—In this infrequent affection there is history of more sudden invasion, and pain is an early symptom, even before pressure has become sufficient to occasion dyspnœa. Fever is likely to be present, and is an early symptom, whereas when it exists in aneurysm it is apt to be late, after the sac has begun to exert pressure on the bronchus or lung with phthisical symptoms. In abscess, moreover, there is not likely to be the change in the vascular sounds or the production of new ones as occurs in aneurysm. The disease may arise at any age and in either sex, showing no predilection for the male sex.

(3) *Pulsating empyema* may simulate an aneurysm when an *empyema necessitatis* forms in close proximity to the base of the heart. It is, however, exceedingly rare, and may occur in children as well as in adults. The history and examination of the lungs ought to clear up the nature of the case. Should a circumscribed empyema in immediate contiguity to the heart display bulging and pulsation as well as dulness, it may occasion considerable difficulty of correct diagnosis, but ought at length to be diagnosed by exclusion, if not by history and physical signs indicative of its real nature.

Other diseases producing signs in the aortic area—i. e., dilatation of the ascending arch associated with aortic regurgitation, stenosis of the aortic ostium, and sclerosis of the ascending arch—may and have been mistaken for aneurysm. In the case of the first mentioned a positive differential diagnosis is sometimes extremely difficult, when the regurgitation occurs in the male past middle age, but as a rule pressure-effects are absent. Thrill and systolic murmur may in cases of stenosis give rise to suspicion of aneurysm, but error may ordinarily be avoided by study of the history, age, the second sound, the position and size of the heart, and the characters of the pulse. Sclerosis of the aorta may occasion a systolic bruit and ringing second sound very suggestive of aneurysm, but does not occasion pressure-effects noted in aneurysm. In all these three affections the subsequent progress will probably clear up the case.

Pulmonary tuberculosis, fibrosis and retraction of the lung and throbbing of the aorta sometimes observed in neurotic subjects ought not to occasion material difficulty if due attention is paid to the history, symptoms, and clinical findings.

Formerly the sphygmograph used to be depended on to aid in the detection of thoracic aneurysm, and may in favourable cases afford reliable information, by furnishing a tracing of one or both radials in which the usual characters are wholly wanting, but in many instances it fails to record positive evidence.

Nowadays we are accustomed to resort to the X-ray in all doubtful or suspected cases. The reader is referred for details to the appropriate article in the Appendix.

Prognosis may be said to be extremely unfavourable, for although spontaneous cure through obliteration of a small sac or one with a narrow pedicle sometimes takes place, it is unlikely for such to happen. Furthermore, the results of medical or surgical treatment are not encouraging. The progress of the disease is not of a necessity steadily downward, although such is apt to be the rule. Remissions may occur both in the gravity of subjective symptoms and growth of the sac.

Thoracic aneurysm may run a comparatively rapid course, particularly if the sac develops externally and ruptures, but the disease may persist for years, depending of course upon the size, direction of growth, and physical conditions of the sac. Ten years may be said to be a long period of time for the continuance of thoracic aneurysm, and yet this limit has been reached and even surpassed. Finally, the outlook is influenced largely by the habits, general status, and environment of the individual, the same as in any other form of cardiac or vascular disease.

Modes and Causes of Death.—The fatal termination may be said to occur either from rupture or the direct or indirect effects of pressure. Death from rupture is not the most frequent mode of termination, as shown by Hare's and Holder's figures, previously quoted, according to which it was the cause of death in 289 out of 953 cases. Rupture may take place externally or into any one of the contiguous structures, pericardium, heart, pleural cavity, bronchus, trachea, œsophagus, vena cava, pulmonary artery. In such an event death may be immediate or protracted over a period of hours.

More commonly, life is terminated in consequence of mechanical interference with respiration or circulation and cardiac inadequacy, or the patient succumbs to " aneurysmal phthisis " or general exhaustion and cachexia. Under such circumstances the end may come slowly or suddenly after weeks of slowly progressing loss of strength. The last hours are in many cases fraught with extreme suffering and death is hailed as a blessed deliverer.

Treatment.—The not infrequent post-mortem discovery of the spontaneous cure of thoracic aneurysm by coagulation of the blood within the sac has furnished the hint upon which all therapeutic measures are based that aim at anything more than palliation of symptoms. The accomplishment of this object presupposes certain favouring conditions in the sac itself. In the first place the aneurysm must be of the saccular variety, and in the second it must communicate with the aorta by a narrow opening. Given these essentials, it is possible for clotting within the sac to take place.

If these conditions are not present, there is little or no prospect of cure, and medical skill is powerless to do more than mitigate suffering or furnish advice, which if carried out may retard progress. In the majority of cases, unfortunately, we are compelled to content ourselves with palliative measures and watching the course of the disease.

Our aim should be, however, to effect a cure in every case in which there seems to be such a possibility. Consequently, the first measure to be advised is *rest in the recumbent position*. The object of this plan of management is the reduction in the number and force of cardiac contractions that thereby the flow of blood within the aneurysm may be less swift. Ever since its introduction by Valsalva the value and importance of this measure has been recognised. To be effective the rest must be absolute and must include rest of mind as well as of body. Whatever excites the heart to more rapid and powerful systoles must be avoided, and to attain as complete rest as is necessary, the patient should be clearly instructed concerning its advantages and necessity.

It is also advisable that arterial tension be reduced and the volume of the blood diminished. To this end the diet must be restricted, as was recommended by Tufnell, of Dublin. His dietary was extremely rigid, consisting as it did of 2 ounces of bread and butter with 2 ounces of milk for breakfast and supper alike, while for the midday meal 2 to 3 ounces of meat and 3 to 4 ounces of milk were allowed.

Such a rigid restriction in the amount of food requires for its successful carrying out courage and determination on the part of the patient, and few persons will submit to such an almost starvation diet. It is probable that the daily allowance may be somewhat greater than Tufnell's dietary permitted without destroying the aim of treatment, provided one remembers that if blood-pressure is to be lowered the quantity of fluid allowed must be small. Furthermore, if such management is to accomplish results it must be persevered in for several months or until the aneurysm gives evidence of having diminished in size. While carrying out this or any other mode of treatment the bowels are to be kept freely open that there may be no straining at stool or increase of blood-pressure incident to constipation.

The next plan of management that promises beneficial results is the administration of iodide of potassium. This mode of treatment was at very nearly the same time recommended by Bouillaud and Chuckerbutty, but has been especially advocated by Balfour. It is not advised because of the syphilitic history obtained in most cases of thoracic aneurysm, but for the purpose of influencing the sac in some as yet unknown manner. Balfour is of the opinion that this salt leads to thickening and contraction of the aneurysmal wall, while others believe its beneficial action lies in decrease of blood-pressure and slowing of the heart's action.

Whatever be its *modus operandi*, it is not necessary and it is not advised to prescribe enormous doses, as used to be done, but to administer it in doses of 5, 10, or 15 grains thrice daily, since these moderate doses accomplish exactly as much as do larger ones. The dose of the salt must not be large enough to produce acceleration of the pulse, the rate of which during repose should have been previously determined. The remedy should be continued for many months, and is advantageously combined with rest and a restricted diet.

Testimony is universal that the first and pronounced effect is relief or very considerable amelioration of pain due to the aneurysm. Why this is cannot be said, but there can be no doubt of the empirical fact. This plan of management should be instituted in all cases, yet to promote a cure of the disease favourable conditions of the sort explained above must be present in the tumour.

The foregoing are the simplest measures, and in most instances are likely to accomplish as much as any other of the various plans of management that have been recommended and will now be mentioned.

The *surgical procedures* sometimes employed in the treatment of this formidable complaint are five in number, as follows:

(1) The introduction into the interior of the sac of many feet of fine iron or steel wire, horsehair, catgut, or silk thread. The object of such treatment is the coagulation of the blood in the meshes of this foreign material, wire being preferable to the others. This operation, known as the Moore or Loreta method, has been done a number of times, but not with sufficiently brilliant results to make it a popular mode of treatment. Of the 16 cases collected by White and Gould (Gibson), only 2 were successful, while of the 8 cases of thoracic aneurysm so treated and collected by Hunner prior to 1900 (Osler), all died. The great objection to this method of management is the resulting inflammation and aggravation of the condition.

(2) Electrolysis, which consists in passing a galvanic current through the contents of the aneurysm by means of two insulated needles introduced through the wall of the sac. The points of the needles are to be left uncovered by the insulating material. They must not be in contact when inside the tumour. The electrical current thus applied causes coagulation of the sac contents, and in Gibson's opinion promises well, although the results as yet have not been very satisfactory. It is worthy of trial in suitable instances.

(3) The Moore-Corradi method, which consists in the combination of the two procedures just mentioned. A fine gold, silver, or steel wire is passed into the sac, and then a galvanic current is sent through the wire. This method is said to have yielded satisfactory results in a few instances. It has been performed by Burresi and Hershey. Of 17 cases of thoracic aneurysm thus treated prior to 1900 only 3 were successful. According to Hun-

ner, this method is not devoid of the following dangers: (1) embolism; (2) the formation of a secondary bulging of the wall of the sac; and (3) obliteration of an artery springing from the wall of the aneurysm.

(4) The scratching of the inner surface of the sac-wall with the point of a thoroughly sterilized needle, a method said to have been introduced by Macewen (Gibson). After the integument has been carefully sterilized an aseptic needle is passed through into the aneurysm until its point comes in contact with the internal surface at the opposite side. The needle may then be left *in situ* to be moved about and made to scratch the lining of the sac by the pulsations of the aneurysm, or the surgeon may irritate the wall by moving the point of the needle about first in one place and then in another, but without removing the instrument. If the needle is left *in situ*, it should not be allowed to remain for longer than twenty-four to thirty-six hours. This method is simple, said to be safe, and to promise well.

(5) The subcutaneous injection of a 1-per-cent solution of pure white gelatin in normal salt solution. This method was introduced by Lancereaux in 1896, and by him was highly praised. At first a 2-per-cent solution was employed, but at the suggestion of Huchard was reduced to half this strength as being safer.

The gelatin solution should be carefully filtered and sterilized under pressure at the temperature of 120° C. Two hundred or 250 cubic centimetres of this 1-per-cent solution at a temperature of about 100° F. are to be very slowly injected into the loose subcutaneous tissue of the thigh or abdomen, after which the patient is to be kept perfectly quiet.

Injections should be repeated every six to eight days until 20 in all have been given. The objections to this plan of treatment are the intense pain and sometimes local and general reaction that follow. In the case of the patient treated in this manner by Dr. Carl Beck, and previously mentioned in these pages, the temperature rose to 101° F. or thereabouts after the injections.

Cures have been reported in France, but in this country, so far as I know, the results have been unsatisfactory. It may be tried in desperate cases; but so many difficulties and dangers attend its use, that it is not likely to become widely employed. Among the dangers is the risk of sepsis or tetanus, since 10 per cent of commercial gelatin is said to contain germs, especially the tetanus bacillus. Moreover, not many patients will be found willing to bear the pain from the injections and the subsequent febrile reaction. Finally, of the cases in which this treatment has been tried, but a small percentage has shown really encouraging results. The use of gelatin in this manner does not appear to increase the coagulability of the blood, and since the action is as yet not understood, it has been suggested that the remedy be given by the mouth as food, 15 grammes being consumed daily.

When one considers the pathology of thoracic aneurysm, the great internal pressure to which the wall of the sac may be subjected in cases in which the mouth of the aneurysm is a wide one, and usually the advanced stage of the process when the individual applies to the surgeon for relief, it is not strange that failure, or at best only amelioration, of symptoms follows any attempt at a cure.

The most that can be done in the great majority of cases is to mitigate the patient's distress. If the iodide of potash does not relieve the pain, recourse must be had to opium in some form. Subcutaneous injections of morphine are the best, since they not only rid the sufferer of his pain for a time, but they also lessen his sense of dyspnœa and promote sleep. I have not yet prescribed heroin in a case of aneurysm, but think it ought in the dose of one-twelfth grain not only to prove efficient against the cough, but should diminish the sense of dyspnœa.

Venesection is highly recommended for relief of venous congestion and to decrease blood-pressure for a time, and thereby the dull pain arising from pressure within the sac. Only a few ounces, 3 to 5, should be taken at a time, since it may have to be frequently repeated, and the abstraction of too much would only serve to weaken the patient without doing more good than do the few recommended.

When the sac is external and large, it is said to minister to the patient's comfort to have him wear an elastic bandage over the tumour (Osler). It certainly ought to lessen the tension to which the integument and thoracic parietes may be subjected, and thereby mitigate pain. In some cases it may be necessary to protect the tumour against violence from external blows by having the patient wear a shield of thin metal or woven wire strapped to his chest.

The diet of these sufferers should be light, even though they are not placed at complete rest, and they should never be allowed to become constipated, since straining at stool is sure to prove harmful. They should take a daily laxative, and now and then, when blood-pressure becomes too high, they should receive a sharp purge from calomel.

They should be informed of the dangerous nature of their malady and be warned of the risk attending severe physical efforts, excitement, excesses, etc.

There are times when from cardiac inadequacy digitalis or one of its congeners may appear indicated, but one should remember that such agents are likely to injure rather than benefit the aneurysm. Consequently if such a remedy is called for, it should be administered with caution and its effects should be carefully watched.

When at length it is plain that the end is near, and, as it approaches, suffering is intense, I am of the opinion that the physician is warranted in the free administration of morphine injections to promote euthanasia. I certainly should not hesitate under such circumstances to inject a dose that would hasten the patient's death. I know of an instance in which this was done to prevent the terrible shock to the friends that was sure to follow the impending rupture of a large external sac.

APPENDIX

MECHANICAL DEVICES AS AIDS TO DETERMINING CARDIAC DISEASE

THE X-RAY

PERCUSSION and auscultation are not entirely satisfactory methods of examining the heart, for the reason that thick, rigid parietes, pulmonary emphysema, or other conditions may prove sources of error. Much depends also on the skill of the examiner or on his delicacy of hearing, so that it is quite common for two or more examiners to obtain results that do not wholly agree. When, therefore, the Roentgen-ray came into use it was quite naturally hoped it would furnish a reliable means of detecting diseased conditions in the heart.

Accordingly, considerable work along this line has been done both in Europe and this country. As a result of such investigations we now know that the X-ray is in many cases a valuable aid to the diagnosis of internal diseases, but cannot altogether replace other and older means of investigation. This is pre-eminently true of cardiac disease.

Francis P. Williams, of Boston, is a particularly diligent investigator with the X-ray, and it is to his elaborate paper in the Philadelphia Medical Journal of January 6, 1900, that I am indebted for much of what is here stated. Percussion of the cardiac area was made by Williams and his friends in a large series of cases both healthy and diseased, and after the limits of deep-seated dulness had been carefully marked out on the bare skin the results thus obtained were compared with those of the X-ray by means of the fluorescent screen. The conclusion Williams arrived at was that the fluoroscope is a much more trustworthy means of judging of the size of the heart. He found that in normal hearts the discrepancies between percussion and the fluoroscope were not so marked as when the heart was either undersized or oversized, and that the greater the enlargement of the organ over the normal, the less frequent is the error by percussion, although the more pronounced is such error when made.

He furthermore discovered the X-ray to be a more precise method of determining the shape and position of the heart. Thus Williams found that in one case, in which the situation of the apex-beat and the results of percussion led him to conclude that the heart was hypertrophied, the X-ray showed the organ to be merely displaced downward so as to lie transversely. Transpositions are also discovered by means of the fluorescent screen more certainly than by percussion. This was brought out very clearly in cases of left-side pleuritic effusions.

Congenital malformations are stated to be capable of diagnosis by the X-ray, and by this means patency of the ductus arteriosus has been determined. It also enables one to diagnosticate a pericardial effusion, as is well illustrated by the figure opposite kindly furnished me by W. C. Fuchs, who took the skiagraph from which the cut has been made (Fig. 118). Cardiac contractions can be observed and differences in size between systole and diastole noted, particularly in cases of valvular incompetence.

The value of the X-ray in the diagnosis of aortic aneurysms has been repeatedly proved. Williams finds that certain aneurysms can be more surely detected by this means than by any other mode of examination. It enables one to determine their location and extent and whether or not the tumour is increasing in size. Finally, if the aneurysm is situated at the left, it is best seen from behind, while those at the right of the heart show best from the front. Although it is possible for even skilled observers to commit error by incorrectly interpreting *normal pulsations* seen by aid of the fluoroscope, still there can be no doubt of the positive value of the X-ray in this class of cases.

To sum up, it may be stated that aside from the detection and study of aneurysms the real practical value of the X-ray in cardiac disease lies in its greater accuracy in determining the size of the heart in general, enlargement of any of the chambers, displacements and transpositions and certain obscure congenital malformations. Even if it could replace percussion and auscultation, which

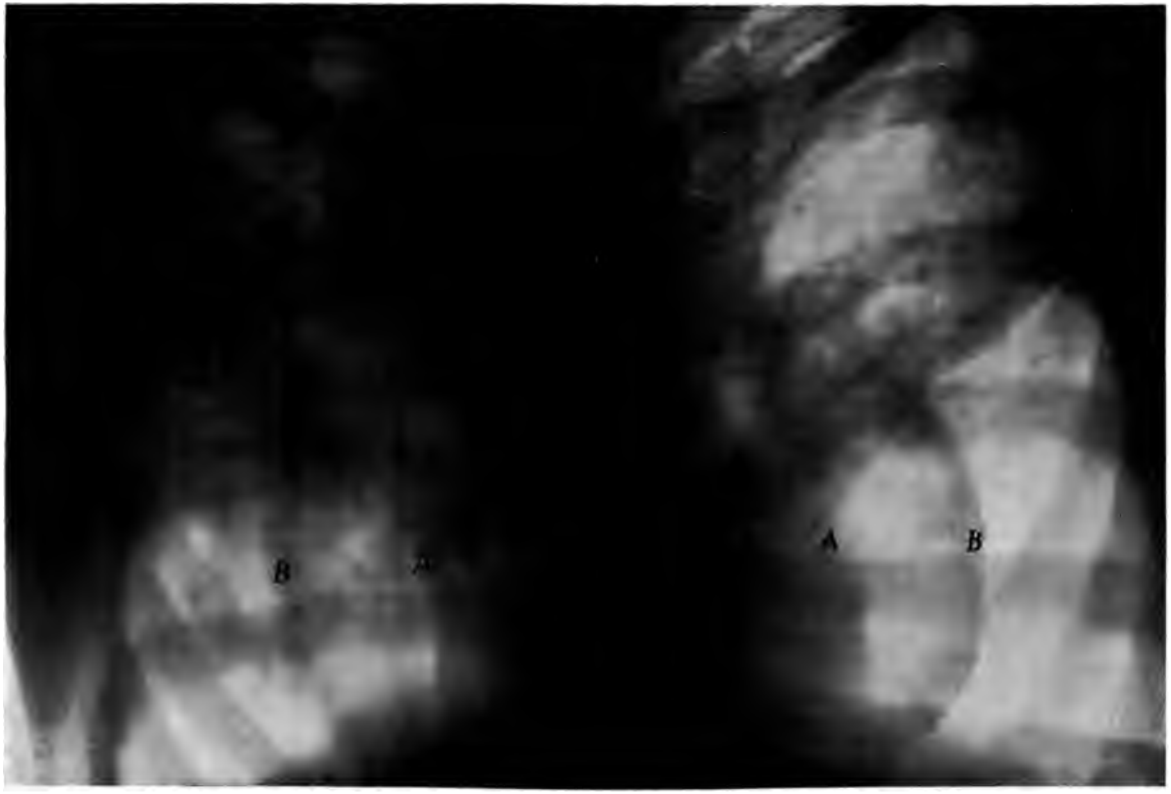


Fig. 118.—Skiagraph of Chest showing Tuberculosis of Right Apex and Tuberculous Pericarditis with Effusion. *AA*, outline of heart; *BB*, outline of distended pericardium.

it cannot, its lack of portability would preclude the possibility of its supplanting older methods.

THE SPHYGMOGRAPH

The sphygmograph is at the same time one of the most useful and most useless of the instruments used in clinical medicine. If used as a routine in his practice by the observing physician it will exceed in value the feeling of the pulse by the fingers, which it should supplement and not supplant. The educated tactile sense, which is always quickly and easily available, can appreciate nearly everything which the sphygmograph can show, and some features which this instrument is unable to delineate, but the impressions cannot be intelligently described and are evanescent. On the contrary, the sphygmograph, which is not always at hand nor readily applicable, can graphically show nearly everything that the finger can detect and some characteristics which this member cannot appreciate, and the results may be preserved for deliberate study, comparison, future reference, and exhibition to others.

The clinician will be able to do good work with any one of the standard sphygmographs, but he can use with the greatest facility, and can interpret most readily and accurately the tracings made by the instrument with which he is most familiar. My own preference is for Dudgeon's sphygmograph, which, because of its portability and adaptability, readily lends itself to the exigencies of all kinds of practice.

In the practical application of the sphygmograph certain elementary rules must be followed, but the whole secret of success in manipulating the instrument lies in placing and maintaining the metal pad upon the artery in such manner as to give the greatest possible amplitude to the excursions of the lever. The wrist band should be elastic; the pad properly placed; the tension correctly adjusted; the pressure gauged to give the greatest amplitude to the writing lever. In adjusting and maintaining the instrument in proper position it is essential that the operator should rely, mainly, upon his fingers and not upon mechanical appliances. Facility in the use of the sphygmograph can only be attained by practice.

It may be noted that the most convenient strips of paper which can be used are made from ordinary heavy writing paper, cut thirty-one thirty-seconds

of an inch wide, and blackened with smoke from burning camphor. The best varnish for preserving the tracings is the ordinary sandarac varnish used by dentists, suitably thinned by the addition of absolute alcohol.

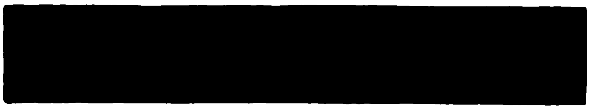


FIG. 119.—FROM A HEALTHY MAN, FORTY-FIVE YEARS OF AGE.

Tracings of the normal pulse in health vary infinitely in their characteristics, and no two are ever exactly alike. The above sphygmogram (Fig. 119) may be considered fairly typical of the pulse in the healthy middle-aged adult.

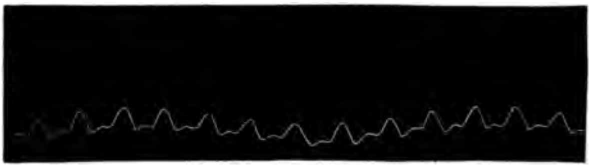


FIG. 120.—FROM A WOMAN, AGED FORTY-FOUR, DURING AN ATTACK OF PAROXYSMAL TACHYCARDIA. PULSE, 196 PER MINUTE.

Between the extreme frequency of the pulse in paroxysmal tachycardia and the remarkable slowness of bradycardia lies a wide gap which is filled by the rapid pulses of infectious fevers, the varying pulses of health and the slow pulses of age, some of the intoxications, etc. (Figs. 120 and 121).

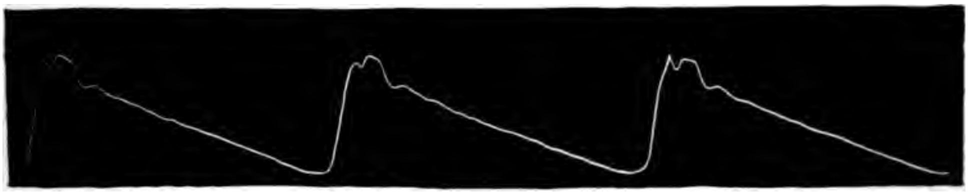


FIG. 121.—FROM A MAN, AGED TWENTY-EIGHT, WITH RECURRENT BRADYCARDIA. PULSE, 25 PER MINUTE.

The sustained arterial tension as shown in the pulse varies within wide limits. The lowest tension is found in some of the acute infections—e. g., general gonorrhœal or pneumococcal, in which there occurs, very early, profound capillary paresis. In many of these cases the powerful left ventricle throws the blood forcibly into the arteries and through the capillaries with practically no resistance, as shown in the following tracing (Fig. 122).

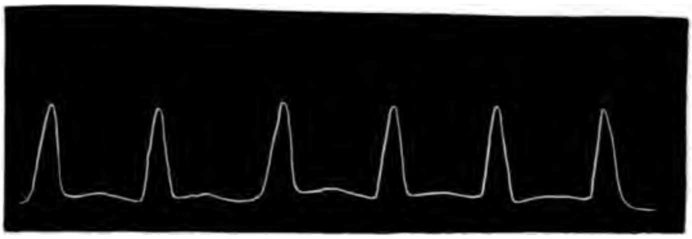


Fig. 122.—From a Man, Aged Twenty-five, with Acute General Gonorrhœal Infection.

In some of these cases the capillary and arteriole vaso-motor reflexes respond energetically to an unnatural stimulus, and the dicrotic pulse of every grade is the result, an example of which is given in Fig. 123. The hyperdicrotic pulse, as shown in Fig. 124, is so often seen in hæmorrhages accompanied by nervous excitement—e. g., in hæmoptysis—that it may be considered somewhat distinctive.

Fig. 123.—From a Man, Aged Forty, with Declining Typhoid Fever.

In aortic regurgitation the powerful left ventricle vigorously propels a large volume of blood into nearly collapsed arteries, quickly and widely distending them, but the flow of blood through the capillaries, during and immediately following the systole, and the reflux of blood through the open valve, the instant ventricu-

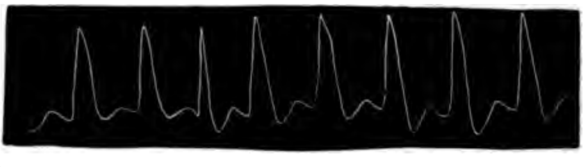


Fig. 124.—Hyperdicrotic Pulse from a Woman, Aged Thirty-five, after Twelve Hours Recurring Hæmoptysis. Pulse, 135.

lar action ceases, as quickly reduce the arterial tension, and the typical pulse of this condition is the result, as shown in Fig. 125.

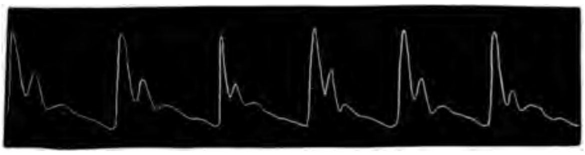


FIG. 125.—FROM A WOMAN, AGED TWENTY-FIVE, WITH MODERATE AORTIC INSUFFICIENCY, WELL COMPENSATED.

In this connection it should be remembered that, other conditions being equal, the pulse will be less frequent and approximate the normal in character, or more frequent and with exaggeration of the distinctive characteristics, according to the degree of valvular incompetency. With failing compensation, the secondary curves in the line of descent may disappear.

At the other end of the scale we have the initial and sustained high-tension pulses, which are so often the accompaniment of early arteriosclerosis, aneurysms of the aorta, and chronic interstitial nephritis, typical tracings of which are given (Figs. 126 and 127).

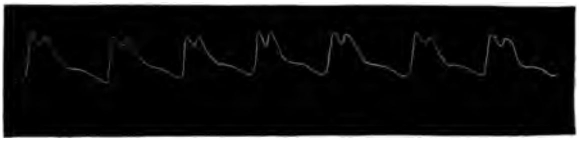


FIG. 126.—INITIAL HIGH-TENSION PULSE, FROM A MAN, AGED FORTY-EIGHT, WITH ARTERIOSCLEROSIS AND A SMALL ANEURYSM OF THE ARCH OF THE AORTA.

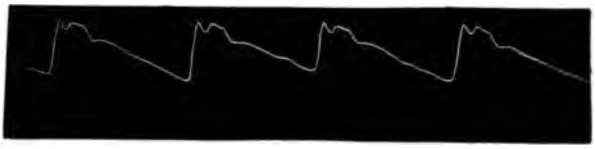


FIG. 127.—SUSTAINED HIGH-TENSION PULSE FROM A WOMAN, AGED SIXTY-THREE, WITH CHRONIC INTERSTITIAL NEPHRITIS.

The following sphygmogram (Fig. 128) may be considered as fairly representing the average in chronic interstitial nephritis, and is typical of those oftenest encountered in this affection.

In this connection it is fair for me to state that, highly as I value the sphygmograph, it is my opinion that its tracings in chronic interstitial nephritis have been accorded, in some quarters, a diagnostic value altogether beyond their merits. This is not to the discredit of the instrument, for it affords the

best practicable means for quickly and conveniently estimating and permanently recording the state of the circulation, cardiac energy, peripheral resistance, arterial resilience, and arterial tension. On the contrary, this fictitious value usually depends upon a faulty appreciation of the infinite variations of the pulse in health and disease, and in the same person at different times.

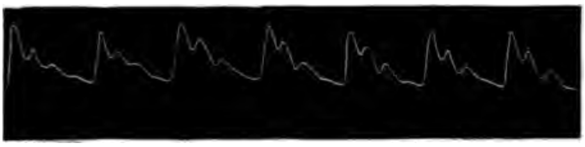


FIG. 128.—FROM A MAN, AGED FORTY-FIVE, WITH CHRONIC INTERSTITIAL NEPHRITIS.

The rhythm of the pulse is very clearly and only satisfactorily shown by the sphygmograph. In health, the rhythm, in every particular, is fairly but not absolutely regular. A moment's reflection upon the physiology of the cardiac cycle and the vaso-motor mechanism should lead us to expect this, and an inspection of any large collection of sphygmograms will confirm the inference. Nevertheless, in ordinary health, the points of difference between the individual pulsations are minute and well within the limits of physiological identity. However, in certain conditions, some of which are understood while others are not, the pulse becomes decidedly and morbidly irregular—arrhythmic. These irregularities may be of almost every conceivable degree and character, some of which are strikingly peculiar.

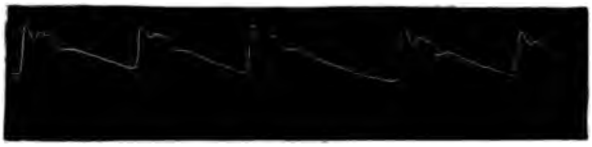


FIG. 129.—FROM A WOMAN, AGED FORTY-FOUR, WITH MILD MYXŒDEMA.

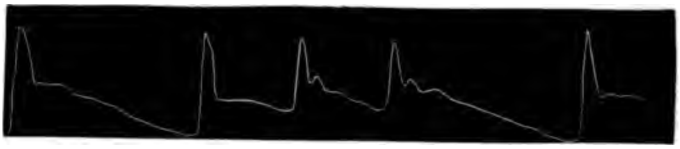


FIG. 130.—FROM A MAN, AGED TWENTY-FOUR, WITH WELL-COMPENSATED MITRAL INSUFFICIENCY.

Thus there may be a marked inequality in the interval between some of the pulsations, as shown in Fig. 129, or beats may be entirely lost, as seen in Fig. 130. It will be found that under these circumstances the line of descent reaches a lower level than it does in the regular pulsations, because the artery has had a longer time in which to empty itself through its distributing channels. In some cases the pulsation is not entirely lost, one or more abortive beats showing in the line of descent, as illustrated in Figs. 131 and 132. Such pulses are denominated bigeminal, trigeminal, etc. It is to be noted that the elevation that marks the abortive pulsation in the bigeminal pulse is located nearer the preceding than the following full beat, and that the second abortive pulsation in the trigeminal pulse lies nearer the first abortive beat than does the latter to the preceding full stroke.

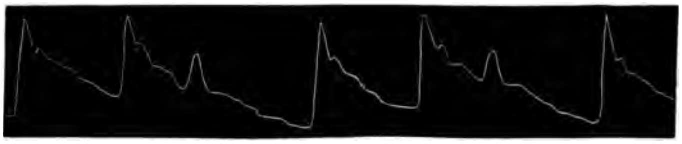


FIG. 131.—FROM A WOMAN, AGED SIXTY-SEVEN, WITH ARTERIOSCLEROSIS AND FAIRLY WELL-COMPENSATED MITRAL INCOMPETENCE.

The arrhythmias thus far mentioned may be irregular in their occurrence, or the prolonged, missed, or abortive pulsations may be repeated at regular intervals. The irregularities of this group may be found rarely in apparent health, and frequently in patients suffering from digestive disturbances, various intoxications—as, e. g., tobacco, renal insufficiency, organic disease of the central nervous system, the vagus and the cardiac ganglia, arteriosclerosis of the coronary arteries, myocardial changes, etc. They occur, therefore, in conditions of no, or varying degrees of danger. They often lead to the discovery of conditions which without such warning might be overlooked. They may, by the strain thrown upon the ventricular walls, lead to dilatation, and relative valvular in-

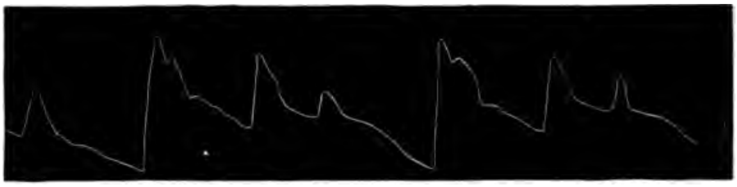


FIG. 132.—FROM A WOMAN, AGED SEVENTY-THREE, WITH ARTERIOSCLEROSIS, AND MITRAL INSUFFICIENCY, FAILING COMPENSATION.

competence and thus become an element of danger. As a matter of fact, however, many persons pass through the greater portion of a long life with such irregularities and without any inconvenience whatever.

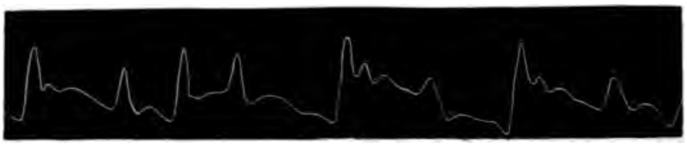


FIG. 133.—FROM A MAN, AGED SEVENTY-THREE, WITH ARTERIOSCLEROSIS, CHRONIC INTERSTITIAL NEPHRITIS, AND MITRAL INSUFFICIENCY, WITH FAILING COMPENSATION. CHEYNE-STOKES RESPIRATION.

In addition to the above arrhythmias we have another group in which the irregularities of the pulse, as shown by tracings, absolutely defy either analysis or description. Such pulses are simply irregularly arrhythmic, and are endless in their variety, as may be seen in the few examples shown (Figs. 133, 134, 135, and 136).

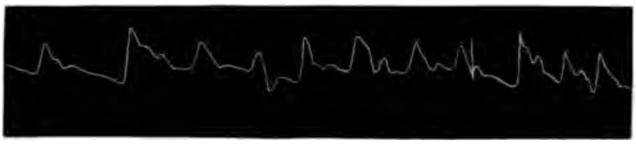


FIG. 134.—FROM A WOMAN, AGED SIXTY-TWO, WITH MITRAL OBSTRUCTION AND REGURGITATION, WITH FAILING COMPENSATION. IRREGULARLY RECURRING DELIRIUM CORDIS.

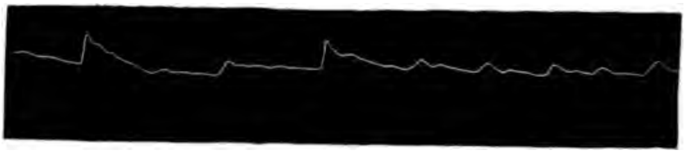


FIG. 135.—FROM A MAN, AGED FIFTY, WITH MITRAL REGURGITATION, LOST COMPENSATION, RELATIVE TRICUSPID INSUFFICIENCY, ASCITES, AND ŒDEMA OF LEGS. LATER COMPARATIVE RECOVERY WITH GOOD COMPENSATION.

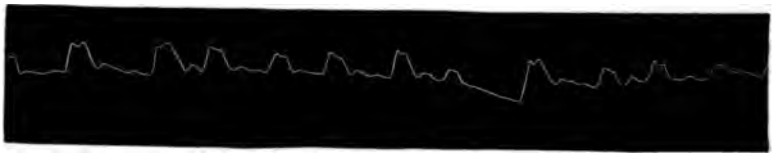


FIG. 136.—FROM A WOMAN, AGED THIRTY-EIGHT, WITH MITRAL OBSTRUCTION AND INSUFFICIENCY, LOST COMPENSATION, AND RELATIVE INCOMPETENCE OF THE TRICUSPID. DELIRIUM CORDIS.

This form of arrhythmia is met with, particularly, in mitral stenosis and incompetence, and in myocardial insufficiency. It is often a late phenomenon in mitral disease. It is of grave, but not necessarily of fatal import, as the lost compensation may be restored, or the weakened myocardium may regain its tone.

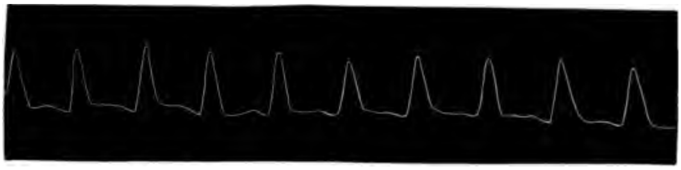


FIG. 137.—FROM A BOY, AGED TWELVE, WITH A PREVIOUSLY NORMAL PULSE, TEN DAYS ILL WITH ACUTE RHEUMATISM, AND ON THE SECOND DAY OF ENDOCARDITIS. NO MURMUR.

In endocarditis the sphygmograph usually furnishes us with diagnostic evidence of valvular involvement several hours or days before murmurs can be heard with the stethoscope. This evidence is shown in a more or less radical change in the character of the pulse, as shown in the above sphygmograms (Figs. 137 and 138).

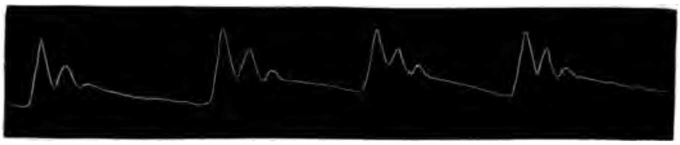


FIG. 138.—FROM THE SAME PATIENT, TWO YEARS LATER, WITH DEVELOPED AORTIC STENOSIS. (CONVALESCENT FROM MUMPS.)

In introducing the above tracings it is proper for me to say that, during the past five years, I have had under observation for one, three, and five years respectively, three cases of aortic stenosis in young persons, from each of whom tracings of a similar character were obtained. In the slighter cases of aortic stenosis the character of the pulse approximates the normal. In this connection a word of caution is due. In using the sphygmograph in cases of considerable or great aortic obstruction the most delicate adjustment of the instrument is required to obtain satisfactory results. It may be said, however, that the greater the care bestowed upon this point the more difficult it will be to produce tracings corresponding to some which have been made classic by more than a generation's text-book currency.

In early pulmonary tuberculosis the pulse is often of a peculiar character, approaching, more or less closely, the infantile type, and the tracings, at this time, possess a distinct diagnostic value.

One of the curiosities of clinical sphygmography is the manifest family resemblance, inherited from the father or mother by their children, often shown in the pulse tracings.

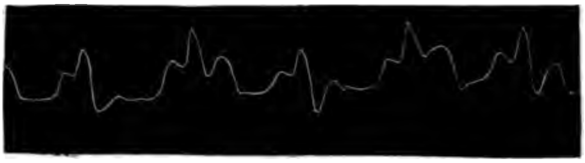


Fig. 139.—Cardiogram from a Girl, Aged Nine, with Mitral Insufficiency.

The sphygmograph may be used to obtain tracings from the heart (an example of which is given in Fig. 139), aneurysms, pulsating veins, pulsating tumours, etc., but the information to be derived from such tracings is not very great.

GAERTNER'S TONOMETER

The examination of the circulation includes the observation of the pulse for what is termed the arterial or blood pressure. This is determined by heart contraction, the peripheral resistance in the arteries and tissues, and the quantity of blood contained in the vessels. Among skilled physicians there is often a difference of opinion in regard to the degree of the arterial pressure, even in general terms, such as hard and soft, while subtile differences are entirely beyond registration, and, to most physicians, beyond perception. Numerous attempts have been made to overcome this difficulty in pulse examinations by means of instruments; if successful, we would then have a more accurate method of comparison of the blood-pressure of individuals, and also of the blood-pressure of the same individual under different conditions. The invention of the sphygmograph was expected to bring accuracy into the subject, but this hope was not realized. Mosso, von Basch, Huerthle, Frey, Oliver, Riva-Rocci, and Hill and Barnard may be mentioned as inventors of such instruments. Of these, the von Basch instrument was the most used up to 1899, since when the Gaertner instrument, on account of greater simplicity of its mechanism, has supplanted it in the hands of many, and has also introduced the practice of taking the arterial pressure to a greater extent than had heretofore been customary. Opinions still vary as to the preferable instrument. James MacKenzie (1902) compares the action of the Hill and Barnard instrument to that of the sphygmograph and regards it unreliable for blood-pressure registration. So also with the Oliver instrument. Jarotzny believes the Hill and Barnard device superior to those of von Basch, Gaertner, etc. Hirsch considers the Gaertner less reliable than the von Basch instrument.

As the latter is at present the chief rival of the Gaertner instrument it may be well to state the principle on which it works. There is a small compressible rubber pelote connected by a rubber tube to a metallic manometer. By pressure with the rubber cylinder (pelote) a suitable artery may be compressed and the amount of necessary pressure at the point of disappearance or reappearance of the pulse on the peripheral side of the instrument is registered on the manometer. The radial artery may be used, but the temporals are usually selected. The sense of touch is required for this instrument, while in the Gaertner method, soon to be described, sight is employed for observing the re-establishment of the circulation; and because in most people the sense of sight is more acute than the sense of touch, the Gaertner instrument requires less practice for its use, and also is believed by many to be more accurate.

Von Basch calls his instrument a sphygmomanometer. Gaertner's tonometer consists of a mercury manometer, a rubber bulb, a "Y" rubber tubing, and a small ring consisting of a metal framework and encased in a rubber envelope, which on inflation stretches inwardly only, and thus compresses the finger that is introduced into the ring. These rings are of different sizes to fit large and small fingers snugly. One end of the "Y" tube is attached to the manometer, another end to the rubber bulb, and the third end to the rubber finger ring; thus pressure made on the bulb transfers itself to the manometer and the rubber ring, and the elastic rubber on the inside of the ring unfolds itself and makes inward pressure in proportion to the pressure put on the bulb. The manometer, being on the same closed tubing, registers the increase or decrease of the pressure. A small clamp is serviceable for compressing the bulb firmly and steadily. The rubber ring is pressed over the first or second phalanx of any finger or the thumb. It is to fit loosely, and is not to rest on a joint. An ordinary small rubber elastic is now rolled from the tip of finger to the rubber ring. This produces an anæmia of the finger. Pressure is now put on the rubber bulb to a degree that is regarded sufficient to maintain the anæmia. This is usually 180 to 200 millimetres of the mercury manometer. The rubber elastic is now pulled off the finger, after which the anæmic appearance continues on account of the constriction of the ring. The pressure on the rubber bulb is now gradually lessened, 5 millimetres at a time. After each diminution, the finger is observed for a few seconds. When the pressure is sufficiently lowered for the arterial pressure to force the blood through the arterioles compressed by the rubber ring, the anæmic finger first shows a few spots of purple congestion, and after a little more reduction of the pressure on the bulb, the finger becomes entirely suffused with the purple colour of congestion, showing that the circulation is re-established. At this point the height of the mercury column is observed on the scale of the manometer. This is the arterial pressure expressed in millimetres of mercury.

One of the principal objections is the small size of the arteries utilized, but Gaertner and others state that the pressure in the digital arteries is the lateral pressure in the volar arch, and that this is probably only 8 or 10 millimetres lower than the pressure in the radial arteries. Another objection is that such small peripheral arteries are more under the influence of the vaso-motor variations than large vessels, and especially subject to local influences. Cold anæmic fingers sometimes must be immersed in warm water before the test can be made.

It is well to follow his instructions for the use of the instrument very closely. The individual may be in the horizontal or upright posture. In the former the pressure is a few millimetres lower than when the person is erect. The manometer must be on a level with the heart. A difference of 10 centimetres in the levels of the heart and manometer produces a change of 7 millimetres in the mercury accordingly. The individual is to breathe regularly. A cough renders the result unreliable on account of the sudden increase of the blood-pressure. The test is not to be repeated on the same finger immediately on account of a possible persistence of an arterial spasm. Thirty seconds will suffice for a test. There is no pain, but at the time of the re-establishment of the circulation the person feels a throbbing and tingling in the finger.

For portability a metallic manometer may be used, but it is not as reliable; it should be frequently compared with the mercury manometer. The advocates of the von Basch method admit the requirement of much more experience and careful manipulation in its use than in that of the Gaertner. The values obtained by both instruments agree fairly, those of the von Basch instrument are probably 8 to 10 millimetres higher; the range of normal blood-pressure under ordinary conditions is from 100 to 160 millimetres of mercury. These limits may be narrowed down to 110 to 135 for the greater number of persons. Constant pressure of 150 to 160 should be regarded suspiciously high. *It is probable that each organism has a mean arterial pressure towards whose maintenance the regulatory mechanism tenaciously strives as soon as a disturbance occurs.* Active influences are numerous. For instance: Posture, food, sleep, physical and mental work, psychical conditions. Several readings should be obtained and the average taken; according to some authorities the lowest reading is the correct blood-pressure, as more causes are active in increasing than lowering the blood-pressure. According to Jellinek the arterial pressure in the fingers of the right side is usually slightly higher than on the left, but Eckart and Hirsch, using von Basch's sphygmomanometer, found the pressure usually higher in the left temporal arteries, and ascribed this to the direct origin of the left carotid artery from the aorta. Hirsch, who prefers von Basch's sphygmomanometer, maintains that the Gaertner instrument registers the blood-pressure 10 to 20 millimetres higher than the von Basch instrument, but admits that in general the values obtained by both instruments agree as to being high, medium, or low.

The high pressures are of special interest on account of being associated with diseases in which many of the threatening symptoms are thought to be due to the high pressure; thus in uræmia and arteriosclerosis pressures of 170 to 240 or more millimetres are the rule. *All observers state that high pressures are frequently found in spite of an apparently soft pulse by palpation;* here, then, as the general accuracy of these instruments cannot be doubted, their value is undeniable. In many illy defined conditions of middle age an unusually high pressure is found, which returns within the normal limits in the course of treatment. Such cases are often described as due to arteriosclerosis or to some intoxication producing increased arterial pressure. In the treatment of nephritis a lowering of the arterial pressure is associated with improvement of the subjective symptoms. In advanced cases of nephritis a sinking of the blood-pressure is considered to presage fatal termination. The influence of muscular efforts on arterial pressure has always been a mooted point. Both moderate increase and decrease have been claimed. This is possibly explained best according to Schott, who finds in such muscular exertions, as wrestling, at first a slight increase of 10 millimetres, but after prolonged dyspnœa a lowering of the pressure from 10 to 25 millimetres. There is certainly not an increase in the pressure directly relative to the amount of muscular exertion (Kornfeld). A cold bath in health as well as in fever increases the blood-pressure from 10 to 15 millimetres. A bath at 104° reduces the pressure slightly; so also do hot air and electric light sweat baths. Exceptions are occasionally found. There is an increase with digitalis, ether, and camphor, but this is less marked, and very frequently absent in fever. In fever the cold bath alone may be relied upon to increase the pressure (Mercandino with the Riva-Rocci instrument); psychic excitement regularly increases the blood-pressure from 10 to 20 millimetres, hence first examinations often are too high. In neurasthenia an increase of 10 to 20 millimetres is so common that Federn, Kraus, and Heim regard it a sign of diagnostic value, especially in children. In hæmatemesis and hæmoptysis there occurs a slight increase of arterial pressure on the second and third days; in acute fevers there is sometimes a slight rise, sometimes a slight sinking of the blood-pressure. The fever does not seem to have a constant influence, but, rather, the blood-pressure varies on account of other factors occurring in the course of the fever. For phthisis a constant low pressure indicates progression, but in the early stages there is usually little change; as the disease progresses the pressure sinks on account of the diminution in the peripheral resistance (Burckhardt-Hensen). There is a diminution in anæmia, cachexia, sleep, and acute cardiac weakness. A pressure of 60 millimetres is considered very grave.

Gaertner states that his instrument registers mean and not maximal pressure. This is not accepted by all. However this may be, we must remember that the actual blood-pressure can be obtained only by the introduction of a cannula into an artery, as has been done in the course of operations by Albert, who found the pressure to be in the anterior tibial artery between 100 and 160 millimetres of mercury; Kuhe-Wiegand during uræmia, 155 millimetres in a radial artery; Faiore in the femoral and brachial arteries, 120 and 110 millimetres; but such direct methods are out of the question for ordinary clinical purposes. The Gaertner instrument, as well as all the other instruments, are influenced by the resistance of the tissue of the artery and the surrounding structures. Von Basch estimates that the resistance of an empty temporal artery to compression is 1 millimetre and that of an atheromatous artery 5 millimetres. The resistance of the soft tissues over the temporal artery is about 6 to 8 millimetres. So after all we are not dealing with figures of the actual pressure within the artery, but with relative figures. If all precautions are observed, these figures may surely be used for comparisons of blood-pressure changes in the same individual with a great degree of reliability; while in comparisons among different individuals much more caution must be observed. Although no defined clinical value can as yet be claimed for these researches except in diseases of high arterial pressure, the necessity for more attention to arterial pressure has become very apparent within the last ten years, as shown by numerous articles appearing on this subject, especially in Germany; and since there are now several instruments giving practical results, we may hope that soon a clearer understanding of the blood-pressure problems will be forthcoming, especially in cardiac diseases and acute fevers, so that both diagnosis and therapeutic indications will become more accurate than has thus far been possible, even with the acumen of the most experienced.

INDEX OF NAMES

Abbott, 804.
Adams, 324, 328, 344, 376, 528, 627, 638, 748.
Albert, 830.
Allbutt, 139, 300, 580, 625, 671, 731, 736.
Andrae, 505.
Aparti, 76.
Apenta, 612.
Arnold, 646, 673, 676, 680.
Arnozan, 376.

Babes, 150.
Bacelli, 397.
Balfour, 27, 263, 353, 496, 501, 541, 640, 801, 810.
Bamberger, 43, 153, 638.
Banholzer, 694.
Banting, 608.
Barié, 19, 366, 368, 374.
Barnard, 826.
Barsdorff, 779.
Basch, 826.
Bauer, 43, 46, 57, 73, 205.
Baumes, 638, 651.
Baumgarten, 764.
Bäumler, 49.
Bax, 629, 636.
Beck, 785, 787, 812.
Beniveni, 505.
Billroth, 766.
Birch-Hirschfeld, 150.
Bizot, 568, 779.
Bodenheimer, 666.
Botalli, 568.
Bouilland, 19, 153, 366, 505, 638, 686, 810.
Bouveret, 730.
Boviard, 376.
Bowles, 730.
Boyer, 627.
Bramwell, 28, 353, 619.
Braun, 766.
Bregmann, 740.
Breid, 692.
Breitung, 81, 85.
Breschet, 681.
Broadbent, 9, 62, 99, 119, 142, 265, 269, 308, 361, 368, 486, 494.
Brochard, 766.
Brotroem, 680.
Browiez, 668.
Browne, 779, 794.
Brunton, 445, 658.
Budd, 153.
Burckhardt-Hensen, 830.
Burresi, 811.
Busse, 6.

Cabot, 698.
Campbell, 250.
Calon, 87.
Cavafy, 730.
Cayley, 150.
Cejka, 79.
Chalmers, 653.
Chambers, 43, 781.
Charcot, 628, 748.
Chatin, 45, 766.
Chauveau, 22.
Cheyne, 237, 531, 537, 539, 550, 616, 632.
Christophe, 108.
Chuckerbutty, 810.
Church, 666.
Clark, 197.
Collet, 766.
Collin, 57.
Cornil, 150.
Corradi, 811.
Corrigan, 298.
Corvisart, 505, 575.
Colton, 730.
Councilman, 741.
Credé, 171.
Crisp, 779.
Cruveilhier, 376.
Culture, 195.

Curschmann, 656.
Curtin, 445.

Davidson, 22.
Davis, 424.
Dessy, 150.
Dieulafoy, 794.
Doehle, 764.
Dorsch, 686.
Dreschfeld, 150, 156, 192.
Drummond, 35, 36, 778.
Duchex, 76.
Duckworth, 137.
Dudgeon, 816.
Duroziez, 286, 305, 311, 399, 498, 690.

Eberth, 182.
Ebstein, 6, 11, 47, 73, 78, 506, 608, 625.
Eckart, 829.
Edes, 627.
Edmunds, 730.
Edwards, 171.
Eichhorst, 81, 139, 567.
Einhorn, 470.
England, 444.
Eppinger, 769, 775, 780.
Erb, 748.
Esmarch, 767.
Eulenberg, 638.
Evans, 112, 161, 359, 542, 700.
Ewart, 72, 75, 77, 81, 674.

Fagg, 153.
Faiore, 831.
Farquharson, 730.
Federn, 730.
Fenger, 293, 376, 379.
Fenwick, 133.
Féré, 690.
Fiessinger, 760.
Figaroli, 76.
Flexner, 42, 45, 156.
Flint, 306.
Forlanini, 376.
Fothergill, 422.
Foxwell, 28, 353.
Fraenkel, 150, 743, 757.
Fraentzel, 19, 321, 526, 534, 544, 558, 568, 744, 773.
François-Franck, 19.
Franz-Josef, 612.
Frazer, 662.
Freund, 512.
Frey, 826.
Friedreichs, 120, 638.
Fuchs, 816.
Fuller, 153.
Fütterer, 460.

Gaertner, 571, 615, 629, 753, 826.
Gairdner, 262, 557, 639, 653.
Galen, 505.
Geigel, 22.
Gerhardt, 254, 730, 772, 779.
Gibson, 28, 119, 139, 195, 343, 403, 640, 648, 666, 690, 694, 730, 760, 779, 804, 811.
Gilbert, 150.
Gintrac, 638.
Givadinovitch, 45.
Goldflam, 766.
Goodhart, 254.
Gorges, 783.
Grave, 714, 727.
Grob, 625.
Gutch, 565.
Guttman, 638.
Guy, 251, 255, 269.

Hadden, 766.
Halberton, 628.
Hale, 482.
Hall, 498.
Hampeln, 534.
Handford, 353.
Hanford, 27.
Hanot, 209.
Hare, 130, 781, 808.
Harris, 99, 104.
Harrison, 590.
Hartell, 678.
Harvey, 741.
Hasenfeld, 156, 744.
Haskin, 170.
Hayden, 139, 153, 269.
Hayem, 506.
Heberden, 637, 654, 655.
Heidman, 116.
Heim, 830.
Hektoen, 116, 669.
Herrick, 356, 361, 364.
Herringham, 730.
Hershey, 811.
Hertz, 676, 678, 680.
Heubner, 742, 764.
Hill, 826.
Hirsch, 829.
Hirschfeld, 506.
His, 627.
Hochhaus, 748.
Hodgson, 775, 779.
Hoffman, 627, 635, 658, 660.

Holder, 130, 781, 808.
Holmes, 168, 794, 799.
Holt, 376.
Hope, 505.
Hornkohl, 6, 8.
Houston, 289, 481, 489, 692.
Howard, 156.
Huchard, 505, 555, 628, 632, 638, 644, 656, 742, 760, 813.
Huerthle, 826.
Hüfler, 470.
Hunner, 811.
Hunter, 637, 647.
Hustedt, 248, 270, 308, 341, 354.
Hutterbrenner, 617.

Janeway, 327.
Jaquet, 627.
Jarotzny, 827.
Jellinek, 829.
Jenner, 637.
Johnson, 68, 294.
Jorn, 470.
Josephson, 70.
Josseraut, 150.
Juda, 779.

Kaczorowski, 358.
King, 344, 672.
Klebs, 150, 157.
Klein, 668.
Klemperer, 195.
Knaggs, 666.
Koehler, 376, 378, 666.
Koenig, 752.
Kolisko, 696.
Kornfeld, 830.
Krannhals, 377.
Kraus, 830.
Krehl, 555, 570, 694.
Krester, 150.
Kreysig, 638.
Kuhe-Wiegand, 831.
Kussmaul, 99, 102, 117, 120, 686, 688, 702, 769.

Laennec, 123, 505, 638, 695.
Lajard, 137.
Lancereaux, 139, 638, 764, 812.
Lancisi, 775.
Landois, 638.
Latham, 639.
Lebert, 153.
Lees, 87.
Leger, 760.
Legg, 673.
Leube, 35, 78, 83, 260.
Leudet, 356, 364.
Lewis, 34.
Leyden, 407, 411, 506, 535, 600, 638.
Lion, 150.
Lobstein, 738.
Loeffler, 157.
Lorence, 288.
Loreta, 811.
Lorry, 637.
Lovewell, 196.

MacCallum, 669.
Macewen, 812.
Mackenzie, 421, 826.
Maguire, 12, 697.
Maier, 769.
Malassez, 694.
Malpighi, 775.
Massa, 505.
Massip, 82.
Mayer, 377.
Meckel, 686.
Mercandino, 830.
Merck, 481.
Miller, 141.
Minor, 436.
Mitchell, 482.
Mollet, 509.
Moore, 811.
Morgagni, 637, 738, 775.
Moritz, 692.
Mosquera, 191.
Mosso, 826.
Mouillé, 695.
Mracek, 139.
Munnely, 730.
Murchison, 169.
Murri, 623.
Musser, 657.

Naunyn, 27.
Netter, 150.
Neusser, 181.
Nothnagle, 638, 731, 759.

O'Byrne, 279.
Oertel, 454, 608, 611.
Oliver, 797, 826, 827.
Ormerod, 43.
Orth, 139, 766.
Ortmann, 766.
Osler, 38, 137, 151, 156, 505, 525, 567, 625, 639, 645, 673, 678, 686, 690, 719, 721, 769, 780, 785, 793, 796, 811.
Otis, 331, 333.

Parr, 741.
Parry, 637.
Pässler, 512.
Paul, 376.
Pawlowski, 671, 676, 679.
Peacock, 153, 686.
Pellatau, 769.
Pembrie, 623.
Perez, 121.
Peter, 638.
Petit, 666.
Philips, 664.
Pick, 100, 123.
Pins, 80.
Pirogoff, 671.
Pitcairn, 42.
Pleischl, 57.
Popoff, 260.
Potain, 19.
Powell, 191, 197, 643, 653, 658.
Poynton, 43, 49, 62, 184, 509.
Preble, 46, 260, 794.
Prentis, 627.
Probsting, 730.
Proust, 676, 680.
Prudden, 144, 150.
Przewoski, 668.
Purcer, 150.

Quain, 706.
Quincke, 28, 298, 301.

Radizewsky, 260, 534, 589.
Recklinghausen, 668, 674, 678.
Redtenbacher, 674, 678, 680.
Reeder, 638.
Regnard, 433, 509, 625, 630.
Rehlmann, 752.
Renaut, 668.
Rendu, 377.
Renvers, 696.
Richardson, 658.
Ricord, 664.
Riegel, 349, 625.
Rinsenna, 377.
Riva-Rocco, 826, 830.
Roberts, 42, 44, 49, 60, 72, 87, 99, 102, 121, 593.
Rokitansky, 254, 276, 506, 686, 695, 738, 769.
Rolleston, 674, 680.
Romberg, 169, 171, 506, 508, 511, 516, 558, 577, 599, 600, 609, 625, 638, 675, 688, 692, 694, 702, 718, 741, 748, 752, 759, 765, 769, 770.
Roque, 766.
Rosenbauch, 77, 150, 164, 328, 506, 518, 526, 620, 638.
Rosenstein, 1.
Rotch, 70, 76, 78, 96.
Rougnon, 633, 637.
Roux, 150.
Rumpf, 661, 755.
Russell, 27, 353.

Samways, 251, 269.
Sansom, 7, 17, 28, 78, 192, 252, 267, 277, 445, 618, 625, 798.
Savart, 22.
Scarpa, 775.
Schlesinger, 766.
Schmaltz, 510.
Schmidt, 778.
Schott, 328, 331, 593.
Schroetter, 683, 759, 772, 775.
Sée, 638.
Semmola, 664.
Seneca, 637, 738.
Sewall, 17, 19, 433.
Shattuck, 77, 96.
Sibson, 43, 49, 66, 76, 683.
Simpson, 689.
Siredi, 376, 760.
Smith, 361.
Sommerbrodt, 559.
Southey, 492.
Stadelmann, 623.
Stange, 674, 680.
Starck, 696.
Starr, 440.
Steele, 300.
Stein, 506.
Sternberg, 766.
Stoelker, 702.
Stokes, 237, 324, 328, 417, 505, 528, 532, 536, 537, 550, 567, 602, 616, 627, 632, 748, 775.
Streeter, 440.
Sturges, 44, 184.
Stybr, 377, 378.
Sweninger, 608.

Talamon, 731.
Tanchon, 666.
Tedeschi, 668.
Teissier, 253.
Thoma, 567, 743, 752, 756, 760, 775, 778.
Thorne, 9.
Tice, 382.
Tiedemann, 638.

Traube, 264, 618, 638.
Tripier, 628.
True, 32.
Tuchzek, 730.

Unverricht, 623.

Vaughn, 197.
Vendeler, 764.
Veronese, 512.
Vesalius, 775.
Vierordt, 5, 11, 693, 756.
Villy, 511.
Virchow, 143, 150, 738, 773.

Walker, 178.
Walsh, 57, 139.
Walshe, 602, 640, 683, 798.
Watson, 730.
Weber, 765, 766, 772.
Webster, 284, 292.
Weichselbaum, 150.
Weigert, 764.
Weirdermann, 766.
Weiss, 766.
Welch, 209, 671, 680.
Welles, 329, 631.
Wells, 136, 733.
Wessner, 471.
West, 731.
Wharry, 617.
White, 811.
Wilks, 99.
Willigk, 666.
Williams, 141, 579, 584, 595, 815.
Wing, 55.
Winge, 143.
Winiwarter, 766.
Wood, 680.
Worcester, 697.
Wunderlich, 153, 668.
Wyssokowitch, 144.

Yung, 273.

Zeissl, 765.
Zenker, 506.
Ziegler, 101, 765.
Ziemssen, 83, 279, 417, 639, 671.

INDEX

Aberrant cords, 30.
Abortion, causing acute endocarditis, 180.
Adherent pericardium, 99;
  Broadbent's sign in, 119;
  Friedreichs' sign in, 120;
  Kussmaul's sign in, 120.
Adhesions, chronic mediastinopericardial, 102;
  formation of, in chronic pericarditis, 103.
Age, influence of, in mitral stenosis, 254;
  in valvular lesions, 407.
Air hunger, 157.
Alcoholism, in acute endocarditis, 47;
  in aortic regurgitation, 280.
Amyl, nitrite of, in angina pectoris, 658.
Aneurysm, congenital, 769;
  cases of, 61, 746.
Aneurysm, of thoracic aorta, 775;
  associated with expectoration and cough, 784;
  auscultation in, 802;
  cases of, 779, 794, 799, 804;
  diagnosis of, 804;
  dyspnœa in, 783;
  electrolysis in, 811;
  etiology of, 777;
  injection of gelatin in, 787;
  of gelatin and salt solution, 812;
  inspection in, 800;
  morbid anatomy of, 775;
  morphine in, 813;
  pain in, 782;
  palpation in, 801;
  percussion in, 802;
  physical signs in, 800;
  prognosis in, 808;
  symptoms of, 781;
  syphilis in, 778;
  treatment of, 809;
  tuberculosis in, 808.
Angina pectoris, 637;
  aconite, not used in, 660;
  amyl nitrite in, 658;
  anodynes in, 660;
  brandy in, 660;
  cases of, 645, 652, 653;
  chloroform and ether in, 658;
  diagnosis of, 654;
  digitalis in, 662;
  etiology of, 640;
  nitroglycerin in, 658;
  opium in, 658, 659;
  pathology of, 640;
  prognosis in, 657;
  strophanthus in, 662;
  syphilis in, 646;
  treatment of, 658.
Angina pseudo-pectoris, 719.
"Angina-sclero-tabagique," 648.
Anodynes, 88.
Antistreptococcus serum. (See Serum.)
Antitoxin, in acute endocarditis, 193;
  in acute myocarditis, 515.
Aorta, stenosis of (see Stenosis);
  thoracic, aneurysm of, 775.
Aortic regurgitation (see Regurgitation):
  stenosis (see Stenosis).
Aortitis, acute, 759;
  etiology of, 760;
  inspection in, 761;
  in measles, 760;
  morbid anatomy of, 159;
  nitroglycerin in, 762;
  palpation in, 761;
  percussion in, 761:
  physical signs in, 762;
  in pneumonia, 760;
  prognosis in, 762;
  in scarlatina, 760;
  strychnine in, 762;
  symptoms of, 760;
  treatment of, 762.

Apnœa, in Cheyne-Stokes respiration, 615.
Applications, cold (see Ice-Bag);
  hot, in acute endocarditis, 190.
Area, aortic, 25;
  cardiac, 25;
  mitral, 26;
  pulmonary, 25;
  tricuspid, 26.
Arrhythmia, in chronic endocarditis, 214.
Arterial system, diseases of, 738.
Arteries, congenital smallness of, 773;
  diagnosis of, 774;
  prognosis in, 774;
  symptoms of, 773;
  treatment of, 774.
Arteriosclerosis, 738;
  bronchitis, chronic, resulting from, 749;
  calomel in, 757;
  cases of, 746, 753;
  diagnosis of, 751;
  digitalis in, 757;
  etiology of, 741;
  jalap in, 757;
  morbid anatomy of, 739;
  nitroglycerin in, 757;
  physical signs in, 750;
  prognosis in, 754;
  strophanthus in, 757;
  symptoms of, 745;
  syphilis in, 742.
Arteritis, acute, 762;
  diagnosis of, 763;
  digitalis in, 762;
  inspection in, 763;
  morbid anatomy of, 762;
  palpation in, 763;
  physical signs in, 763;
  prognosis in, 763;
  symptoms of, 762;
  treatment of, 763.
Arteritis, syphilitic, 764;
  diagnosis of, 766;
  etiology of, 765;
  morbid anatomy of, 764;
  prognosis in, 766;
  symptoms of, 765;
  treatment of, 766.
Artery, cerebral, rupture of, in hypertrophy of left ventricle, 574;
  pulmonary, stenosis of (see Stenosis).
Ascites, in adherent pericardium, 117.
Asthma, cardiac, 237, 613.
Asthma, bromides in, 563;
  in mitral stenosis, 270.
Atheroma. (See Arteriosclerosis.)
Atrophy of the heart, 667;
  diagnosis of, 668;
  etiology of, 667;
  morbid anatomy of, 667;
  prognosis in, 668;
  symptoms of, 668;
  treatment of, 668.
Atropine, in Cheyne-Stokes respiration, 623;
  in valvular lesions, 500.
Attack, neuroses, treatment of, 727.

Bacilli. (See Micro-organisms.)
Bacteria. (See Micro-organisms.)
Baths, hot, evil effect of, in valvular lesions, 427.
  in valvular lesions, 427, 466, 503;
  Nauheim, 110, 115, 464, 503, 592;
  saline, in valvular lesions 466;
  Turkish, 552.
Belladonna, in pericarditis, 88.
Benign endocarditis. (See Endocarditis.)
Bloodletting, in dilatation of heart, 591.
Blue baby, of congenital heart disease, 692, 701.
Bradycardia, 624;
  diseases associated with, 625;
Breathing, Cheyne-Stokes, diseases in which, observed, 617.
Bright's disease, in pericarditis, acute, 45;
  chronic, in Cheyne-Stokes respiration, 617;
  in myocarditis, chronic, 539;
  in regurgitation, mitral, 237.
Broadbent's sign in adherent pericardium, 119.
Bronchial disorders, in valvular lesions, 407.
Bronchitis, in pericarditis, acute, 47;
  in pericarditis, chronic, 102.

Caffeine, in tachycardia, 736;
  in valvular lesions, 432.
Calcification in pericarditis, chronic, 101.
Calomel, in arteriosclerosis, 757;
  in pericarditis, chronic, 125;
  with effusion, 89;
  in valvular lesions, 432, 448, 491, 493.
Cancer, of the myocardium, 666.

Cardiac asthma (see Asthma);
  neurosis, 703;
  pain, 718.
Catarrh, bronchial, in mitral lesions, 407.
Cathartics, in dilatation, 592;
  in endocarditis, chronic, 202;
  in valvular lesions, 492.
Cheyne-Stokes respiration, 617;
  case of, 622;
  in diphtheria, 617;
  morphine in, 623;
  in pneumonia, 617;
  prognosis in, 622;
  treatment of, 623.
Chills and fever, in suppurative pericarditis, 72.
Chloralamide, in chronic myocarditis, 563.
Chloral hydrate, in valvular lesions, 501.
Chloralose, in valvular lesions, 501.
Chlorosis, in mitral insufficiency, 597.
Chorea, in acute endocarditis, 153, 154, 155.
Cirrhosis, atrophic hepatic, differentiated from adherent pericardium, 122;
  renal, leading to tricuspid regurgitation, 345.
Climate, change of, in valvular lesions, 432.
Clothing, in valvular lesions, 425, 476.
Codeine, in pericarditis, 88, 91.
Compensation, imperfect, in valvular lesions, 435.
  lost, 478;
  perfect, 413, 414;
  prevented, in chronic pericarditis, 105.
Congenital aneurysm. (See Aneurysm.)
Congenital diseases of the heart, 686;
  case of, 698;
  diagnosis of, 701;
  etiology of, 689;
  inspection in, 695;
  morbid anatomy of, 686;
  morphine in, 693;
  palpation in, 696.
Congenital smallness of arteries. (See Arteries.)
Congestion, abdominal viscera, in aortic and mitral regurgitation, 397;
  cerebral, in mitral regurgitation, 237.
Congestion, chronic pulmonary, 231;
  venous, 397.
Cords, aberrant, 30.
Corpulent, cardiac inadequacy of the, 599.
Cough, in aneurysm of thoracic aorta, 784.
Cusp, rupture of, in aortic regurgitation, 278.
Cyanosis, in acute endocarditis, 171;
  in aortic stenosis, 335;
  in dilatation, 585;
  in tricuspid regurgitation, 347.

Death, mode and causes of, in aneurysm of thoracic aorta, 808;
  in regurgitation, aortic, 307;
  mitral, 247;
  pulmonary, 374;
  tricuspid, 354;
  in stenosis, aortic, 340;
  mitral, 270;
  pulmonary, 388;
  tricuspid, 364;
  sudden, in syphilis of the myocardium, 665.
Deglutition, painful, in dry pericarditis, 49.
Delirium, in mitral stenosis, 270.
Devices, mechanical, as aids to determining diseases, 815.
Dextrocardia, acquired, 682;
  diagnosis of, 684;
  etiology of, 683;
  inspection in, 685;
  morbid anatomy of, 682;
  palpation in, 684;
  percussion in, 684;
  prognosis in, 685;
  symptoms of, 684;
  treatment of, 685;
  tuberculosis in, 681.
Dextrocardia, congenital, 681;
  case of, 681;
  symptoms of, 681.
Diathesis, rheumatic, in valvular lesions, 406.
Digitalis in arteriosclerosis, 757;
  in arteritis, 762;
  in angina pectoris, 662;
  in dilatation, 591;
  in endocarditis, acute, 189;
  in endocarditis, chronic, 202;
  in fatty heart, 611;
  in functional disorders, 709;
  in hypertrophy, 575.

Digitalis in mitral insufficiency, 598;
  in myocarditis, acute, 516;
  in myocarditis, chronic, 552, 564;
  in pericarditis, chronic, 126;
  in pericarditis, dry, 54;
  in pericarditis, with effusion, 83;
  routine administration, objectionable, 89;
  in pneumopericardium, 136;
  in regurgitation, aortic, 288, 290;
  in regurgitation, mitral, 225;
  in stenosis, aortic, 326;
  in stenosis, mitral, 272;
  in stenosis, tricuspid, 359;
  in Stokes-Adams disease, 636;
  in syphilis, of myocardium, 665;
  in tachycardia, 736;
  in valvular lesions, 394, 430, 480;
  warning in regard to use of, 497.
Dilatation, 576;
  baths in, 592;
  bloodletting in, 591;
  blue mass in, 591;
  cases of, 582, 584, 593;
  cathartics in, 592;
  cyanosis in, 584;
  diagnosis of, 586;
  digitalis in, 591;
  etiology of, 577;
  influenza in, 577;
  inspection in, 585;
  morbid anatomy of, 576;
  morphine in, 593;
  nitroglycerin in, 591, 593;
  palpation in, 585;
  percussion in, 585;
  physical signs in, 585;
  prognosis in, 587;
  resistance exercises in, 592;
  rheumatism in, 401, 429;
  secondary to pericarditis, 101;
  strychnine in, 591;
  symptoms of, 580;
  treatment of, 590.
Diphtheria, in bradycardia, 625;
  in Cheyne-Stokes respiration, 617;
  in endocarditis, acute, 156;
  in myocarditis, acute, 508, 511;
  in pericarditis, acute, 46;
  in tachycardia, 732.
Diphtheritic endocarditis. (See Endocarditis.)
Disorders, functional. (See Functional Disorders.)
Dropsy, in hydropericardium, 130;
  in myocarditis, chronic, 530, 563.
Dropsy, in regurgitation, mitral, 219, 236, 245;
  cause of, in tricuspid regurgitation, 348;
  in regurgitation, tricuspid, 351;
  in valvular lesions, 470.
Drugs, use of, in valvular lesions, 430.
Duroziez's sign, in aortic regurgitation, 305.
Dyspepsia, chronic, in bradycardia, 625.
Dyspnœa, in aneurysm, aortic, 783;
  in endocarditis, acute, 171;
  in regurgitation, mitral, 238;
  in stenosis, mitral, 257.

Electrolysis, in aneurysm of thoracic aorta, 811.
Embolism, septic, in acute endocarditis, 158, 172, 184.
Emphysema, in hypertrophy, 570.
Endarteritis obliterans, 766;
  diagnosis of, 768;
  etiology of, 767;
  morbid anatomy of, 766;
  prognosis in, 768;
  symptoms of, 767;
  treatment of, 769.
Endocarditis, acute, 143;
  abscess in, 156, 181;
  aconite, not used in, 189;
  alcohol in, 192;
  alkalies in, 187;
  applications in, hot, 190;
  associated with myocarditis, 157;
  associated with pericarditis, 101, 157;
  bacillus of diphtheria in, 151, 156;
  of influenza in, 151, 156;
  of typhoid fever in, 151;
  bacteria, pyogenic, in, 149, 156;
  blister in, 188;
  brandy in, 191;
  bromides in, 189;
  camphor in, 191;
  in cancer, 156;
  cases of, 158, 164, 170;
  chorea in, 153, 154, 155;
  course of, 163;
  cyanosis in, 171;
  diagnosis of, 163;
  diagnosis, differential, from typhoid fever, 182;
  digitalis in, 189;
  dyspnœa in, 171;
  emboli in, 172;
  ether in, 191;
  etiology of, 143.

Endocarditis, acute, hemiplegia in, 158, 184;
  ice-bag in, 189;
  indicated by rheumatic fever, 157;
  infarction in, 158;
  in fœtal life, 143;
  inspection in, 176;
  leucocytosis in, 181;
  in measles, 154;
  morbid anatomy of, 143;
  morphine in, 175;
  opium in, 190;
  oxygen in, 191;
  palpation in, 176;
  in pelvic disease, 155;
  percussion in, 177;
  physical signs in, 176;
  in pneumonia, 186;
  in pyæmia, 156;
  pyrexia in, 171;
  resulting from enteric fever, 154;
  resulting from gall-stones, 156;
  resulting from scarlet fever, 154;
  rheumatism in, 146, 152, 153, 157, 181, 186, 187;
  sepsis in, 170, 193;
  serum, antistreptococcus in, 193;
  in small-pox, 154;
  strophanthus in, 169;
  strychnine in, 169, 191;
  symptoms of, 157;
  in tonsillitis, 156;
  treatment of, 187.
Endocarditis, benign, 143.
Endocarditis, chronic, 199;
  arrhythmia in, 214;
  cases of, 201, 206, 210;
  cathartics in, 202;
  digitalis in, 202;
  etiology of, 201;
  morbid anatomy of, 199;
  nitroglycerin in, 202;
  rheumatism in, 204;
  strychnine in, 202;
  symptoms of, 205;
  syphilis in, 204;
  treatment of, 202.
Endocarditis, diphtheritic, 143.
Endocarditis, malignant, 143.
Endocarditis, mycotic, 143.
Endocarditis, simple, 143, 150, 157.
Endocarditis, ulcerative, 143, 154, 163;
  course of, 172;
  diagnosis of, 179;
  morphine in, 196;
  treatment of, 191.
Endocarditis, vegetative, 143.
Endocarditis, verrucose, 143.
Endocardium, diseases of, 143.
Enteric fever. (See Fever.)
Epilepsy, in mitral stenosis, 212.
Ewart's sign, in pericarditis with effusion, 80, 81.
Exercise, in dilatation, 592;
  resistance, 455, 592;
  in valvular lesions, 414, 454, 502.
Expectoration, in aneurysm of thoracic aorta, 784.

Fatty heart, 599;
  aperients in, 612;
  camphor in, 611;
  case of, 607;
  diagnosis of, 605;
  diet in, 608;
  digitalis in, 611;
  etiology of, 600;
  gentian in, 610;
  gluttony, predisposing to, 601;
  hypophosphites in, 610;
  inspection in, 604;
  iron in, 610;
  morbid anatomy of, 599;
  nitroglycerin in, 611;
  nux vomica in, 610;
  orthopnœa in, 603;
  palpation in, 604;
  pathology of, 599;
  percussion in, 604;
  physical signs in, 604;
  prognosis in, 606;
  strophanthus in, 611;
  strychnine in, 611;
  symptoms of, 602;
  treatment of, 606.
Fever, in pericarditis, dry, 51;
  in pericarditis, with effusion, 89;
  enteric, in endocarditis, 154;
  rheumatic, indicating acute endocarditis, 157;
  scarlet, Cheyne-Stokes respiration in, 617;
  in myocarditis, 522;
  leading to acute endocarditis, 154;
  leading to pericarditis with effusion, 72.
Fever, typhoid, Cheyne-Stokes respiration in, 617;
  diagnosis of, differential, from acute endocarditis, 182;
  in bradycardia, 625;
  in endocarditis, acute, 151.

Fever, in myocarditis, acute, 508;
  in myocarditis, chronic, 522;
  typhoid, in pericarditis, acute, 46;
  treatment of, in acute myocarditis, 516.
Fibroma, of myocardium, 666.
First rib sign, in pericarditis with effusion, 75.
Fœtal life, acute endocarditis in, 143;
  developmental anomalies in, 690;
  perforate interventricular sæptum in, 688.
Fomentations, in pericarditis, 88.
Food, in valvular lesions, 428.
Fragmentation of myocardium, 688.
"Fremissement cataire," in mitral stenosis, 259.
Friedreich's sign in adherent pericardium, 120.
Friction-sounds. (See Sounds.)
Functional disorders, 703;
  cases of, 705, 714;
  digitalis in, 709;
  neuroses in, 703;
  strophanthus in, 711;
  strychnine in, 709;
  tuberculosis in, 716.

Gangrene of foot, in arterial thrombosis, 676;
  of leg, in mitral regurgitation, 238.
Gastritis, chronic, in chronic myocarditis, 551.
Gelatin, injection of, in aneurysm, 787.
Germs. (See Micro-organisms.)
Glonoin, in valvular disease, 432, 446.
Gluttony, inducing fatty heart, 601.
Goître, exophthalmic, hypertrophy in, 570;
  tachycardia in, 715.
Gonorrhœa, in acute endocarditis, 154, 181;
  in acute myocarditis, 508.
Gout, in mitral stenosis, 254;
  in regurgitation, aortic, 280, 296.

Habits, in valvular lesions, 410, 420, 476.
Hæmopericardium, 130.
Hæmophilia, in acute pericarditis, 47.
Heart, area, aortic, 3;
  mitral, 4;
  pulmonic, 3;
  tricuspid, 3.
Heart, atrophy of. (See Atrophy.)
Heart, auscultation of, 12;
  deep boundaries of, 6;
  dilatation of (see Dilatation);
  diseases of, congenital, 686;
  disorders of, functional, 703;
  enlargement of, 5;
  fatty (see Fatty Heart);
  hypertrophy of (see Hypertrophy);
  location of, 1;
  musical, notable example of, 30;
  position of, attempt to fix, 2;
  relation of, to anterior thoracic wall, 1;
  size of, how ascertained, 5;
  valve lesions of the right, summary of physical signs of, 389;
  vessels and valves, position of, 3.
Heart sounds. (See Sounds.)
Hemiplegia, embolic, in acute endocarditis, 158, 184.
Heroin, in pericarditis with effusion, 88.
Home surroundings, in valvular lesions, 410.
Hydropericardium, 103, 127, 131;
  diagnosis of, 129;
  dropsy in, 130;
  etiology of, 128;
  inspection in, 128;
  morbid anatomy of, 127;
  palpation in, 128;
  percussion in, 129;
  physical signs in, 128;
  prognosis in, 129;
  pyrexia in, 129;
  rheumatism in, 129;
  symptoms of, 128;
  treatment of, 129.
Hyperæmia, chronic, 116.
Hypnotics, in valvular disease, 500.
Hypophosphites, in fatty heart, 610;
  in valvular lesions, 448.
Hypertrophy of the heart, 565;
  aconite, not used in, 575;
  diagnosis of, 572;
  digitalis in, 575;
  etiology of, 568;
  following emphysema, 570;
  inspection in, 571;
  morbid anatomy of, 565;
  palpation in, 571;
  percussion in, 571;
  physical signs in, 571;
  prognosis in, 574;
  symptoms of, 570;
  treatment of, 575.

Ice, in paroxysmal tachycardia, 736.
Ice-bag, in acute endocarditis, 189;
  in pericarditis, 87, 89.
Illnesses in valvular lesions, 429.
Inadequacy, cardiac, of the corpulent, 599.
Incompetency, cardiac, 555.
Individual tendencies, in chronic endocarditis, 206.
Infarction, in acute endocarditis, 158;
  in acute myocarditis, 514.
Infection, affecting valves, 185.
Influenza, in dilatation, 577;
  in acute endocarditis, 151, 156;
  in fatty heart, 600;
  in myocarditis, chronic, 522, 551;
  in paroxysmal tachycardia, 732.
Injection of gelatin in aneurysm, 787;
  of gelatin and salt solution, 812.
Insomnia, in mitral regurgitation, 237;
  in mitral stenosis, 256.
Insufficiency, mitral. (See Mitral Insufficiency.)
Insurance, life. (See Life Insurance.)
Iron, in fatty heart, 610;
  in insufficiency, mitral, 597;
  in myocarditis, acute, 517;
  in valvular lesions, 448.

Kidneys, in arteriosclerosis, 741;
  in endocarditis, acute, 150, 158, 185;
  in endocarditis, chronic, 203;
  in myocarditis, acute, 514;
  in pericarditis, acute, 45;
  in pericarditis, chronic, 112;
  in regurgitation, mitral, 238;
  in valvular disease, 490.
Knowledge of lesion, effect on patient, 411.
Kussmaul's sign, in adherent pericardium, 120.
Kyphoscoliosis, resulting in hypertrophy of right ventricle, 570.

Lesions, valvular. (See Valvular Lesions.)
Leucocytosis, in acute endocarditis, 181.
Life insurance, relation to, of prognosis in valvular disease, 412.
Lipoma, of myocardium, 666.
Liver, cirrhosis of, 102, 156;
  in dilatation, 584;
  in endocarditis, acute, 156, 171, 185.
Liver, in pericarditis, chronic, 100, 102, 105, 109, 117, 122;
  in pericarditis, dry, 63;
  in pericarditis, with effusion, high position of, 70;
  in regurgitation, aortic, 288;
  in regurgitation, mitral, 219, 233, 238;
  in regurgitation, tricuspid, 347, 350;
  in stenosis, mitral, 257, 268;
  in valvular disease, 405, 464;
  pseudo-, Pick's pericarditic, 123.

Maculæ tendiniæ, 101.
Magnesia, sulphate of, in valvular disease, 447, 492.
Malignant endocarditis. (See Endocarditis.)
Marriage, in valvular disease, 422.
Massage, in chronic myocarditis, 559.
Measles, resulting in acute aortitis, 760;
  resulting in acute endocarditis, 154;
  resulting in acute pericarditis, 46.
Mechanical devices as aids to determining disease, 815.
Mediastinitis, associated with pericarditis, 101.
Mediastinopericarditis, 103, 104, 105;
  Perez's sign in, 121.
Medicinal agents, in valvular disease, 444.
Micro-organisms, in abscess, 508;
  in blood, 181;
  in dilatation, 577;
  of diphtheria, 151, 156;
  in endocarditis, acute, 144, 149, 150;
  gas forming, 133;
  of influenza, 151;
  in myocarditis, acute, 508;
  in pericarditis, acute, 40, 42;
  in penumopericardium, 133;
  pyogenic, 149;
  of tuberculosis, 42, 332;
  of typhoid fever, 151.
Mitral insufficiency, relative, 594;
  chlorosis in, 597;
  diagnosis of, 596;
  digitalis in, 598;
  etiology of, 594;
  iron in, 597;
  nitroglycerin in, 594;
  pathology of, 594;
  physical signs in, 596;
  prognosis in, 597;
  in rheumatism, 595;
  symptoms of, 596.

Mitral insufficiency, treatment of, 597.
Mitral regurgitation (see Regurgitation);
  stenosis (see Stenosis).
Mode and causes of death. (See Death.)
Moderator bands, 30.
Morbus ceruleus, in pulmonary stenosis, 385.
Morphine, in aneurysm of thoracic aorta, 813;
  in Cheyne-Stokes respiration, 623;
  in congenital diseases, 693;
  in dilatation, 593;
  in endocarditis, acute, simple, 175;
  in endocarditis, ulcerative, 196;
  injection of, in aneurysm, 814;
  in myocarditis, chronic, 533, 561;
  in pericarditis, dry, 69, 88, 91;
  in pneumopericardium, 135;
  in regurgitation, aortic, 288, 290, 316;
  in stenosis, aortic, 333;
  in stenosis, mitral, 272;
  in Stokes-Adams disease, 635;
  in valvular disease, 446, 481, 499.
Murmurs, accidental, 26, 32;
  differential diagnosis of, 34;
  not accompanied by secondary changes, 35;
  detection of, 13;
  endocardial, 21;
  exocardial, 36;
  musical, 29;
  pericardial, effect of pressure on, 59;
  transmission of, 25. (See also Sounds.)
Muscle, papillary, degeneration of, a cause of mitral insufficiency, 541.
Mycotic endocarditis. (See Endocarditis.)
Myocarditis, acute, 505;
  antitoxin in, 515;
  associated with endocarditis, 157;
  diagnosis of, 514;
  digitalis in, 516;
  diphtheria in, 508, 511;
  etiology of, 508;
  infarction in, 158;
  iron in, 517;
  micro-organisms in, 508;
  morbid anatomy of, 506;
  palpation in, 514;
  percussion in, 514;
  in physical signs in, 514.
Myocarditis, prognosis in, 515;
  pulse in, 511, 513;
  rheumatism in, 508, 513, 515;
  scarlatina in, 508, 516;
  small-pox in, 508;
  strophanthus in, 516;
  symptoms of, 510;
  treatment of, 515;
  in typhoid fever, 508, 516.
Myocarditis, chronic, 518;
  atropine in, 562;
  baths in, Turkish, 552;
  brandy in, 561;
  Bright's disease, associated with, 539;
  bromides in, 563;
  bronchitis, acute, in, 551;
  camphor in, 561;
  cases of, 526, 531, 540, 541;
  chloralamide in, 563;
  diagnosis in, 122, 547;
  digitalis in, 522, 564;
  dropsy in, 530, 563;
  etiology of, 522;
  gastritis, chronic, in, 551;
  influenza in, 522, 551;
  inspection in, 543;
  massage in, 559;
  morbid anatomy of, 519;
  morphine in, 533, 561;
  nitroglycerin in, 553, 560;
  palpation in, 543;
  percussion in, 544;
  physical signs in, 543;
  pneumonia in, 551;
  prognosis in, 549;
  pulse in, 529;
  rheumatism in, 522, 541;
  strophanthus in, 553, 561;
  strychnine in, 553;
  symptoms of, 526;
  treatment of, 551;
  typhoid fever in, 522.
Myocardium, cancer of, 666;
  degeneration of, in chronic pericarditis, 101;
  diseases of, 505;
  fibroma of, 666;
  fragmentation of, 668;
  lipoma of, 666;
  segmentation of, 668.
Myocardium, syphilis of, 663;
  diagnosis of, 664;
  digitalis in, 665;
  etiology of, 663;
  iodides in, 665.

- Myocardium, syphilis of, mercury in, 665;
  - morbid anatomy of, 663;
  - prognosis in, 665;
  - symptoms of, 664;
  - treatment of, 665.

- Nephritis, in acute pericarditis, 44.
- Neuroses, 703, 717, 731;
  - diagnosis of, 724;
  - etiology of, 722;
  - pain in, 728;
  - pathology of, 703;
  - prognosis in, 726;
  - symptoms of, 704;
  - treatment of, 727.
- Nitroglycerin, in angina pectoris, 658;
  - in aortitis, acute, 762;
  - in arteriosclerosis, 757;
  - in dilatation, 591, 593;
  - in endocarditis, chronic, 202;
  - in fatty heart, 611;
  - in mitral insufficiency, 594;
  - in myocarditis, chronic, 553, 560;
  - in pseudo-angina pectoris, 728;
  - in regurgitation, aortic, 288, 314, 316;
  - in stenosis, aortic, 332, 333;
  - in stenosis, mitral, 272;
  - in Stokes-Adams disease, 635;
  - in tachycardia, 715;
  - in valvular diseases, 442, 444, 446, 488, 498.

- Occupation, effect of, in valvular diseases, 409, 419, 476.
- Œdema, in mitral stenosis, 256;
  - in valvular diseases, 495;
  - digitalis in, 495.
- Orthopnœa, in fatty heart, 603;
  - in pericarditis with effusion, 67.
- Oxygen, in acute endocarditis, 191;
  - in Stokes-Adams disease, 635.

- Pain, in aneurysm of thoracic aorta, 782;
  - attack of, in neuroses, 728;
  - cardiac, 718;
  - in pericarditis, dry, 49.
- Palpitation, in cardiac neuroses, 727.
- Paroxysmal tachycardia, 730;
  - features of, 732.
- Pathogenesis, of thrombi, 674.
- Pathology of angina pectoris, 640;
  - of fatty heart, 599;
  - of mitral insufficiency, 594;
  - of neuroses, 703.
- Pathology of Stokes-Adams disease, 627;
  - of tachycardia, 731.
- Pectoris, angina (see Angina Pectoris);
  - pseudo-angina, 719.
- Percussion, "abgedämpfte" method, 7;
  - auscultatory, or stethoscopic, 8;
  - palpatory, 10.
- Perez's sign in chronic mediastino-pericarditis, 121.
- Pericarditis, acute, 37;
  - abscess in, 47;
  - alcoholism in, 47;
  - Bright's disease in, 45;
  - bronchitis in, 47;
  - cancer in, 47;
  - caries of rib in, 47;
  - cholera in, 46;
  - diphtheria in, 46;
  - erysipelas in, 46;
  - etiology of, 41;
  - measles in, 46;
  - micro-organisms in, 40, 42;
  - morbid anatomy of, 37;
  - nephritis in, 44;
  - peritonæum, diseases of, in, 47;
  - pleuritis in, 47;
  - pneumonia in, 46;
  - purulent form, 40;
  - purpura hæmorrhagica in, 47;
  - rheumatism in, 42, 46;
  - scarlatina in, 46;
  - scurvy in, 47;
  - serofibrinous form, 40, 42;
  - simplest form, 37;
  - small-pox in, 46;
  - strychnine in, 516;
  - suppurative form, 42;
  - tonsillitis in, 44, 47;
  - typhoid fever in, 46;
  - ulcer in, 47;
  - valvular defects resulting from, 49.
- Pericarditis, chronic, 99;
  - baths in, 110, 115;
  - calomel in, 125;
  - cases of, 105, 114, 123;
  - compensation prevented in, 105;
  - course and termination of, 117;
  - diagnosis of, 122;
  - diagnosis, differential, from cirrhosis of liver, 122;
  - digitalis in, 126;
  - diuretin in, 126;
  - etiology of, 103;
  - morbid anatomy of, 100.

Pericarditis, chronic, palpation in, 120;
  percussion in, 121;
  physical signs in, 118;
  prognosis in, 123;
  rheumatism in, 117;
  stasis in, 117;
  strophanthus in, 114;
  strychnine in, 114, 125;
  symptoms of, 104;
  treatment of, 124.
Pericarditis, dry, 48;
  cases of, 50, 52, 61;
  course and termination of, 56;
  deglutition in, painful, 49;
  diagnosis of, 60;
  differential, 60;
  digitalis in, 54;
  inspection in, 56;
  morphine in, 69, 88, 91;
  pain in, 49;
  palpation in, 57;
  percussion in, 57;
  physical signs in, 56;
  pneumonia in, 60;
  prognosis in, 61;
  pyrexia in, 51;
  rheumatism in, 50, 54, 61;
  strychnine in, 53;
  symptoms of, 48;
  hæmorrhagic, 40, 47.
Pericarditis, with effusion, 64;
  anodynes in, 88;
  atropine in, 92;
  belladonna in, 88;
  blister in, 86, 87;
  calomel in, 89;
  cases of, 68, 70, 92;
  chloroform in, 88;
  codeine in, 88, 91;
  course and termination of, 73;
  diagnosis of, 81;
  differential, 82;
  digitalis in, 83;
  fever in, 89;
  "first rib" sign in, 75;
  fomentations in, 88;
  heroin in, 88;
  ice bag in, 87, 89;
  inspection in, 75;
  opium in, 88, 91;
  orthopnœa in, 67;
  percussion in, 76;
  physical signs in, 74;
  prognosis in, 84;
  puncture in, site of, 94, 96.
Pericarditis, resulting from pneumonia, 73;
  with effusion, resulting from scarlet fever, 72;
  rheumatism in, 68, 70, 73, 84;
  sepsis in, 72;
  signs in, "first rib," 75;
  Ewart's, 80, 81;
  Pins', 80;
  Rotch's, 78;
  strychnine in, 91;
  symptoms of, 64;
  treatment of, 86, 90;
  tuberculosis in, 84.
Periarteritis nodosa, 769;
  etiology of, 769;
  morbid anatomy of, 769;
  prognosis in, 770;
  sepsis in, 769;
  symptoms of, 769;
  treatment of, 770.
Pericardium, adherent, 99;
  bacteria in, 45;
  signs in, Broadbent's, 119;
  Friedreich's, 120;
  Kussmaul's, 120.
Pericardium, carcinoma of, 141;
  diseases of, 37;
  perforated by gastric ulcer, 133;
  sarcoma of, 141;
  syphilis of, 139;
  tuberculosis of, 136.
Pick's pericarditic pseudo-cirrhosis of the liver, 123.
Pins' sign, in pericarditis with effusion, 80.
Pleurisy, mistaken for pericarditis, 83.
Pneumonia, in aortitis, acute, 760;
  in bradycardia, 625;
  in Cheyne-Stokes respiration, 617;
  in endocarditis, acute, 185;
  croupous, 154, 156, 181;
  recovery from, 155;
  in myocarditis, chronic, 551;
  in pericarditis, acute, 46;
  in pericarditis, chronic, 103;
  in pericarditis, dry, 60;
  in pericarditis, with effusion, 73, 97;
  in regurgitation, mitral, 224;
  in stenosis, aortic, 341;
  in valvular disease, 440.
Pneumopericardium, 132;
  bacilli in, gas forming, 133;
  brandy in, 135;
  cases of, 133;
  diagnosis of, 135.

Pneumopericardium, digitalis in, 136;
  etiology of, 132;
  inspection in, 134;
  morbid anatomy of, 132;
  morphine in, 135;
  prognosis in, 135;
  resulting from trauma, 133;
  resulting from ulcer, 133;
  strychnine in, 136;
  symptoms of, 133;
  treatment of, 135.
Pregnancy, in valvular defects, 409, 422.
Pressure, effect of, on pericardial murmur, 59.
Pseudo-angina pectoris, 719;
  -cirrhosis of liver, Pick's pericarditis, 123.
Pulmonary artery, stenosis of (see Stenosis).
Pulmonary regurgitation (see Regurgitation);
  stenosis (see Stenosis).
Pulse, capillary, in aortic regurgitation, 301;
  inequality of, in aortic aneurysm, 801;
  inequality of, in mitral stenosis, 257, 259, 260;
  instability of, in acute myocarditis, 511, 513;
  tension of, in chronic myocarditis, 529;
  venous, in aortic regurgitation, 301;
  "water hammer," in aortic regurgitation, 298.
Puncture, site of, in pericarditis with effusion, 94, 96.
Pyrexia, in endocarditis, acute, 171;
  in hydropericardium, 129;
  in pericarditis, dry, 51;
  in regurgitation, tricuspid, 346.

Quincke's sign, in aortic regurgitation, 298.

Regurgitation, aortic, 278;
  alcoholism in, 280;
  cases of, 282, 288, 293, 308, 313;
  cusp, ruptured in, 278;
  diagnosis of, 305;
  digitalis in, 288, 290;
  Duroziez's sign in, 305;
  etiology of, 280;
  gout in, 280, 296;
  inspection in, 288, 290, 316.
Regurgitation, aortic, nitroglycerin in, 288, 314, 316;
  palpation in, 298;
  percussion in, 301;
  physical signs in, 297;
  prognosis in, 306;
  pulse in, 298, 301;
  Quincke's sign in, 298;
  rheumatism in, 289;
  scarlatina in, 309;
  strophanthus in, 288, 291;
  strychnine in, 290, 316;
  symptoms of, 282;
  syphilis in, 284.
Regurgitation, aortic and mitral, combined, 397;
  diagnosis of, 391, 397;
  prognosis in, 398;
  symptoms of, 397.
Regurgitation, aortic, and aortic stenosis combined, 396.
Regurgitation, aortic, and mitral stenosis combined, 393;
  inspection in, 395;
  palpation in, 395;
  percussion in, 395;
  prognosis in, 394, 396;
  symptoms of, 393.
Regurgitation, mitral, 216;
  Bright's disease in, 237;
  cases of, 224, 229, 232, 247;
  congestion in, 237;
  diagnosis of, 245;
  digitalis in, 225;
  dropsy in, 219, 236, 245;
  dyspnœa in, 238, 257;
  etiology of, 252;
  inspection in, 239;
  insomnia in, 237:
  morbid anatomy of, 216;
  palpation in, 239;
  percussion in, 240;
  physical signs in, 239;
  pneumonia in, 224;
  prognosis in, 246:
  resulting in gangrene, 238;
  rheumatism in, 222, 247;
  scarlatina in, 222, 224, 229;
  stasis in, 236;
  strychnine in, 225;
  symptoms of, 223;
  tuberculosis in, 232.
Regurgitation, mitral, and aortic stenosis combined, 396.
Regurgitation, mitral, and mitral stenosis combined, 392.

Regurgitation, symptoms of, 391.
Regurgitation, of pulmonary artery, 772;
  diagnosis of, 772.
Regurgitation, pulmonary, 365;
  case of, 368;
  diagnosis of, 387;
  etiology of, 380.
  morbid anatomy of, 365;
  palpation in, 371;
  percussion in, 371;
  physical signs in, 370;
  prognosis in, 374;
  stasis in, 367;
  symptoms of, 367.
Regurgitation, tricuspid, 343;
  case of, 354;
  cyanosis in, 347;
  diagnosis of, 363;
  dropsy in, 351;
  etiology of, 356;
  inspection in, 349;
  morbid anatomy of, 344;
  palpation in, 350;
  percussion in, 350;
  physical signs in, 349;
  prognosis in, 354;
  pyrexia in, 340;
  resulting from cirrhosis of lung, 340;
  from fibroid phthisis, 346;
  from renal cirrhosis, 345;
  secondary to chronic bronchitis, 346;
  stasis in, 347;
  symptoms of, 347.
Rheumatism, acute, in bradycardia, 625;
  in endocarditis, acute, 146, 181, 186;
  in endocarditis, chronic, 522;
  in myocarditis, acute, 513, 515;
  in pericarditis, acute, 42;
  in pericarditis, chronic, 117;
  in pericarditis, dry, 50, 54;
  in regurgitation, aortic, 289;
  in regurgitation, mitral, 222;
  in stenosis, pulmonary, 377;
  in tachycardia, 732;
  in valvular disease, 401, 429.
Rheumatism, articular, 32, 34;
  in dilatation, 583;
  in endocarditis, acute, 152, 157, 187;
  in hydropericardium, 129;
  in mitral insufficiency, 595;
  in myocarditis, acute, 508.
Rheumatism, articular, in pericarditis, acute, 42;
  in pericarditis, dry, 61;
  in pericarditis, with effusion, 70;
  in regurgitation, mitral, 247;
  in stenosis, pulmonary, 376;
  in stenosis, tricuspid, 356;
  in valvular disease, 441, 479, 485.
Rheumatism, inflammatory, in endocarditis, acute, 153;
  in myocarditis, chronic, 541;
  in pericarditis, with effusion, 68, 73, 84;
  in stenosis, mitral, 274;
  in valvular disease, 436, 441.
Rhythm, gallop or canter, 18;
  a sign of the end, 20.
Rotch's sign, in pericarditis with effusion, 78.

Scarlatina, in aortitis, acute, 760;
  in congenital disease, 698;
  in endocarditis, acute, 154;
  in myocarditis, acute, 508, 516;
  in pericarditis, acute, 46;
  in regurgitation, aortic, 309;
  in regurgitation, mitral, 222, 224, 229;
  in stenosis, mitral, 274;
  in stenosis, tricuspid, 358.
Scarlet fever. (See Fever.)
Sclerosis, 246, 286, 573.
Scurvy, in pericarditis, acute, 47.
Second sound, simulated doubling of, 17.
Segmentation of the myocardium, 668.
Sepsis, in acute endocarditis, 170, 193;
  in periarteritis nodosa, 769;
  in pericarditis with effusion, 72.
Septicæmia, in acute endocarditis, 155, 156.
Serum, anti-streptococcus, in acute endocarditis, 193.
Signs, Broadbent's, in adherent pericardium, 119;
  Duroziez's, in aortic regurgitation, 305;
  Ewart's, in pericarditis with effusion, 80, 81;
  "first rib," in pericarditis with effusion, 75;
  Friedreich's, in adherent pericardium, 120;
  Kussmaul's, in adherent pericardium, 120.

Signs, Perez's, in chronic mediastino-pericarditis, 121;
  Pins', in pericarditis with effusion, 80;
  Quincke's, in aortic regurgitation, 298;
  Rotch's, in pericarditis with effusion, 78.
Simple endocarditis. (See Endocarditis.)
Smallness of arteries, congenital, 773.
Small-pox, in acute endocarditis, 154;
  in acute myocarditis, 508;
  in acute pericarditis, 46.
Sound-friction, intensity of, 59;
  location of the pericardial, 58;
  quality of, 59;
  rhythm of, 58.
Sounds, heart, normal, 13;
  reduplication of, 16, 18;
  second, simulated doubling of, 17.
Spleen, abscess of, in acute endocarditis, 150, 185.
Stasis in pericarditis, chronic, 117;
  in regurgitation, aortic, 297;
  in regurgitation, mitral, 236;
  in regurgitation, pulmonary, 367;
  in regurgitation, tricuspid, 347;
  in stenosis, mitral, 257;
  in stenosis, tricuspid, 358.
Stenosis, of aorta, 770;
  symptoms of, 771;
  treatment of, 772.
Stenosis, aortic, 319;
  cases of, 323, 330, 339;
  cyanosis in, 335;
  diagnosis of, 338;
  digitalis in, 326;
  inspection in, 335;
  morbid anatomy of, 319;
  nitroglycerin in, 332, 333;
  palpation in, 335;
  percussion in, 336;
  physical signs in, 335;
  pneumonia in, 338;
  prognosis in, 339;
  strychnine in, 332;
  symptoms of, 323.
Stenosis, aortic and mitral combined, 392;
  diagnosis of, 392;
  prognosis in, 393;
  symptoms of, 392.
Stenosis, mitral, 249;
  bronchitis in, 256;
  cases of, 253, 263, 270, 273.
Stenosis, mitral, delirium in, 270;
  diagnosis of, 268;
  digitalis in, 272;
  epilepsy in, 212;
  "fremissement cataire" in, 259;
  gout in, 254;
  insomnia in, 256;
  inspection in, 258;
  morbid anatomy of, 249;
  morphine in, 272;
  nitroglycerin in, 272;
  œdema in, 256;
  palpation in, 259;
  percussion in, 260;
  physical signs in, 258;
  prognosis in, 269;
  pulse in, 257, 259, 260;
  rheumatism in, 264;
  scarlatina in, 274;
  stasis in, 257;
  strophanthus in, 272;
  strychnine in, 272;
  symptoms of, 255;
  syphilis in, 254.
Stenosis, mitral, and pulmonary stenosis combined, 387.
Stenosis, pulmonary, 376;
  cases of, 377, 380;
  diagnosis of, 373;
  inspection in, 385;
  morbid anatomy of, 376;
  percussion in, 386;
  physical signs in, 385;
  prognosis in, 374;
  rheumatism in, 376, 377;
  symptoms of, 380;
  tuberculosis in, 380.
Stenosis, of pulmonary artery, 772.
Stenosis, tricuspid, 355;
  cases of, 379, 380;
  diagnosis of, 353;
  digitalis in, 359;
  inspection in, 361;
  morbid anatomy of, 355;
  percussion in, 362;
  physical signs in, 361;
  prognosis in, 364;
  rheumatism in, 356;
  scarlatina in, 358;
  stasis in, 358;
  symptoms of, 357.
Stokes-Adams disease, 627;
  cases of, 628, 630, 632;
  digitalis in, 636;
  etiology of, 627;
  morphine in, 635.

Stokes-Adams disease, nitroglycerin in, 635;
oxygen in, 635;
pathology of, 627;
prognosis in, 635;
symptoms of, 629;
syphilis in, 628;
treatment of, 635.
Strophanthus, in angina pectoris, 662;
in arteriosclerosis, 757;
in endocarditis, acute, 169;
in fatty heart, 611;
in functional disorders, 711;
in myocarditis, acute, 516;
in myocarditis, chronic, 553, 561;
in pericarditis, chronic, 114;
in regurgitation, aortic, 288, 291;
in stenosis, mitral, 272;
in valvular disease, 432.
Strychnine, in aortitis, acute, 762;
in dilatation, 591;
in endocarditis, acute, 169, 191;
in endocarditis, chronic, 202;
in fatty heart, 611;
in functional disorders, 709;
in myocarditis, chronic, 553;
in pericarditis, acute, 516;
in pericarditis, chronic, 114, 125;
in pericarditis, dry, 53;
in pericarditis, with effusion, 91;
in pneumopericardium, 136;
in regurgitation, aortic, 290, 316;
in regurgitation, mitral, 225;
in stenosis, aortic, 332;
in stenosis, mitral, 272;
in tachycardia, 736;
in valvular disease, 439, 443, 445.
Syncope, in aortic regurgitation, 287.
Syphilis, in aneurysm, 778, 795;
in arteriosclerosis, 742;
in angina pectoris, 646;
in endocarditis, chronic, 204;
in regurgitation, aortic, 284;
in stenosis, mitral, 254;
in stenosis, tricuspid, 356;
in Stokes-Adams disease, 628;
Syphilis of the myocardium, 663;
of the pericardium, 139.
Syphilis vs. rheumatism, in aortic stenosis, 388.
Syphilitic arteritis, 764.

Tachycardia, 730;
caffeine in, 736;
diagnosis of, 734;
digitalis in, 736;
diphtheria in, 732.
Tachycardia, etiology of, 732;
ice in, 736;
influenza in, 732;
malaria in, 732;
nitroglycerin in, 715;
pathology of, 731;
prognosis in, 735;
rheumatism in, 732;
strychnine in, 736;
treatment of, 735.
Temperaments, of cardiopaths, 408.
Tendencies, individual, 206.
Terrain cure, in valvular disease, 454.
Thoracic aorta, aneurysm of, 775.
Thoracic cavity, 1.
Thrombi, ball, 674;
bibliography of, 680;
cases of, 676, 680;
diagnosis in, 677;
etiology of, 674;
pathogenesis of, 674.
Thrombi, pedunculated, 674;
prognosis in, 678;
symptoms of, 675;
treatment of, 678.
Thrombosis, arterial, causing gangrene, 676;
venous, in valvular disease, 208.
Tissue, adipose, in syphilis of pericardium, 139.
Tonics, accessory, 499;
cardiac, 441;
nerve, 517.
Tonsillitis, in endocarditis, acute, 156;
in pericarditis, acute, 44, 47.
Tonometer, Gaertner's, 826.
Tricuspid regurgitation (see Regurgitation);
stenosis (see Stenosis).
Tuberculosis, of pericardium, 136;
of lungs, 103.
Tuberculosis, pulmonary, in aneurysm, 808;
in dextrocardia, 681;
in functional disorders, 716;
in pericarditis, with effusion, 84;
in regurgitation, mitral, 232;
in stenosis, pulmonary, 380;
in valvular disease, 406.
Typhoid fever, in pericarditis, 46.
Typhus, in acute myocarditis, 508.

Ulcerative endocarditis. (See Endocarditis.)

Valvular lesions combined, 390;
atropine in, 500.

Valvular lesions, baths in, Nauheim, 427, 464, 503;
baths in, saline, 466;
caffeine in, 432;
calomel in, 432, 448, 491, 493;
cases of, 393, 436, 440, 469, 479, 482, 485;
cathartics in, 492;
chloral hydrate in, 501;
chloralose in, 501;
change of climate in, 432;
clothing in, 425, 476;
compensation imperfect in, 435;
compensation lost in, 478;
compensation perfect in, 413;
complicated with catarrh, bronchial, 407;
with dropsy, 470;
with pneumonia, 440;
with rheumatism, 406;
convallaria in, 432, 497;
diet in, 428, 470;
digitalis in, 394, 430, 480;
drugs in, 430;
exercise in, 414, 454, 502;
exercise, resistance, in, 455;
glonoin in, 432, 446;
habits in, 410, 420, 476;
hæmatics in, 448;
home surroundings in, 410;
hypnotics in, 500;
illnesses in, 429.
Valvular lesions, jalap in, 493;
marriage in, 422;
medicinal agents in, 444;
mercury in, 432;
morphine in, 446, 481, 499;
nitroglycerin in, 442, 444, 446, 488, 498;
occupation in, 409, 419, 476;
œdema in, 495;
pregnancy in, 409;
prognosis in, 401;
rest in, 448, 502;
rheumatism in, 401, 429, 436, 441, 479, 485;
strophanthus in, 432;
strychnine in, 439, 443, 445;
Terrain cure in, 454;
treatment of, 413, 435, 478;
tuberculosis in, 406.
Vegetative endocarditis. (See Endocarditis.)
Verrucose endocarditis. (See Endocarditis.)
Vessels and valves of heart, position of, 3.

Whisky, in acute endocarditis, 192;
in angina pectoris, 660;
in pseudo-angina, 728;
in myocarditis, chronic, 561;
in valvular disease, 493.

(1)

THE END

THE DIAGNOSTICS OF INTERNAL MEDICINE

A CLINICAL TREATISE UPON THE RECOGNIZED PRINCIPLES OF MEDICAL DIAGNOSIS, PREPARED FOR THE USE OF STUDENTS AND PRACTITIONERS OF MEDICINE

By GLENTWORTH REEVE BUTLER, A. M., M. D.

Chief of the Second Medical Division, Methodist Episcopal Hospital; Attending Physician to the Brooklyn Hospital; Consulting Physician to the Bushwick Central Hospital; formerly Associate Physician, Departments of Diseases of the Chest and Diseases of Children, St. Mary's Hospital, Brooklyn, N. Y.; Fellow of the New York Academy of Medicine; Member of the Medical Society of the County of Kings, etc.

224 Illustrations, Five Plates, Twenty-two Charts
8vo, 1061 Pages, Cloth, $.600

"This is a valuable book upon an important subject. The general plan of the work, the arrangement of subjects, the colored plates, the illustrations and diagrams, are alike excellent. The book as a whole is, consequently, a reliable guide for students and practitioners in this very important field of medical practice."—*Journal of Medicine and Science.*

"Works on diagnosis are comparatively numerous at the present time, but each new one has its special features of interest and value. The present volume covers the greater portion of the field of internal medicine in a very satisfactory way, and undoubtedly will be found by many more acceptable than other works. It is particularly strong in its illustrations; these are generally very well selected to illustrate the text, which itself is clear and readable. It is well worth a place in the physician's library."—*Journal of the American Medical Association, Chicago.*

"This volume presents some unusual features, and will find many readers who will appreciate the method by which the subject is treated. The great bulk of the work deals with the evidences of disease, and does so in a most satisfactory manner. The student is taught how to proceed in the examination of his patient, what routine questions are to be asked such as would lead to a preliminary opinion which in turn would suggest certain examinations in detail. The author teaches not only what to do, but how to do it, and his advice and method of diagnosis are to be highly recommended. The smaller details, which only acute observers notice, are pointed out and their significance is weighed. It will be of service as a reference book in suggesting explanations in cases where but one or few symptoms, not pathognomonic, are presented for diagnosis—for each symptom is explained and the ordinary diseases in which it occurs are mentioned, while in the latter part of the book the diseases are treated separately, so that direct suggestions can be immediately considered without consulting another volume. All the modern laboratory methods are well described, the chapters on the blood and stomach contents being especially good, though brief. The thorough modernness of the instruction is well instanced by drawings of the culex and anopheles genera of mosquitoes. The author is evidently fully conversant with the clinical evidences of disease, for his treatment of the chapters on the signs and symptoms which present themselves in the patient is unusually well balanced with reference to their importance. The section on pain is very thorough; the illustrations are profuse and particularly instructive."—*Medical Record.*

D. APPLETON AND COMPANY, NEW YORK

INTRODUCTION TO THE STUDY OF MEDICINE

By G. H. ROGER

PROFESSOR EXTRAORDINARY IN THE FACULTY OF MEDICINE OF PARIS
MEMBER OF THE BIOLOGICAL SOCIETY
PHYSICIAN TO THE HOSPITAL OF PORTE-D'AUBERVILLIERS

AUTHORIZED TRANSLATIONS BY
M. S. GABRIEL, M. D.

WITH ADDITIONS BY THE AUTHOR

8vo. Cloth, $5.00

"Such a work, well conned, provides beginners in medicine with a sure and wide foundation of systematic science on which to base all their future acquirements. Dr. Roger's unique volume is to be heartily commended, not only to would-be physicians but also to that large class of post-graduates who do not cease to be students because they have left college, and would like to know the reasons and relations of things."—*Medical Times, Denver, Col.*

"The work throughout shows an essentially logical method of treatment, and covers a very wide subject in a manner which amply demonstrates the author's extensive acquaintance with medical thought and medical literature. The book can be recommended not only to students, but also to practitioners in medicine, as giving a wide and philosophical view of medicine and its tendencies at the present day."—*Albany Medical Annals, Albany, N. Y.*

"As a *résumé* of recent medical advancement we may expect this volume to occupy a unique place. It is something of a novelty to turn the pages of a medical work which will be most valuable to the profession as a whole, not to the specialist or individual student alone. It is, however, well suited to use as a text-book, and will put students in touch with medicine as an all-embracing science. But to those of the profession—and there are many—who lack opportunity to thoroughly acquaint themselves with the rapid strides which are being made in knowledge of the causation of disease, lesions and reactions of the organism which are discussed under pathological anatomy and semeiology, the work will serve as a substitute for lectures and laboratory experience. Some of the principal chapters deal with the mechanical, physical, chemical, and animate agencies of disease, the general etiology and pathogenesis of the infectious diseases, nervous reactions, disturbances of nutrition, heredity, inflammation, septicemia and pyemia, tumors, cellular degenerations, examination of the sick, clinical application of scientific procedures, diagnosis and prognosis, therapeutics, etc. An immense amount of work is evidenced by the text, and much careful and scholarly research. A book of this kind is needed, and will be particularly appreciated by those who, without undervaluing the importance of laboratory investigations, still think clinical methods and the simpler means of reaching a diagnosis and prognosis should not be forgotten or slighted."—*New England Medical Gazette, Boston, Mass.*

D. APPLETON AND COMPANY, NEW YORK

LANE MEDICAL LIBRARY

To avoid fine, this book should be returned on
or before the date last stamped below.

MAY 2 8 2001

MAY 1 8 2001